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THE PROCEEDINGS

OF THE

SECOND

ALL-INDIA

SANITARY CONFERENCE

HELD AT

MADRAS

November 11th to 16th, 1912.

VOLUME III—RESEARCH.

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Prefatory note.

This volume contains papers on RESEARCH which were read at the Second All-India Sanitary Conference. It has not been found possible to reproduce all illustrations, plans, etc., attached to the various papers, and anyone specially interested should apply to the authors.

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PART I.

TRAVELLING DISPENSARIES.

2.

ALL-INDIA SANITARY CONFERENCE—MADRAS—NOVEMBER 1912.

TRAVELLING DISPENSARIES IN THE UNITED PROVINCES.

BY

Captain H. Ross, I.M.S., Chief Plague Officer, United Provinces.

In 1910 the Hon'ble Colonel C. C. Manifold, I.M.S., Inspector-General of Civil Hospitals, United Provinces, proposed the establishment of four travelling dispensaries to be placed in four districts in the United Provinces, Government allotting a sum of Rs. 6,000 for their upkeep.

These were experimental and their working was very carefully watched, each being under the direct supervision of the Civil Surgeon of the district in which it worked.

Their chief object being to treat malaria and distribute quinine to the sick, they were known as malarial travelling dispensaries and were only sanctioned for a period of six months, July to December, the season when malaria is most prevalent.

Colonel Manifold, with the idea of a great future extension of this form of village medical relief, directed that they should take up the treatment of various forms of general disease from the beginning.

In 1911, on being consulted by His Honour Sir John Hewett, with regard to what measures could be taken to combat the spread of plague which during the cold weather of 1910-11 had raged with great virulence in these provinces, he proposed a large extension of these travelling dispensaries, at the same time laying great stress on the importance of adequate supervision by special touring medical officers.

In carrying out his proposals this was fully recognised and careful attention paid to it.

In his farewell speech to the Legislative Council of the United Provinces, His Honour Sir John Hewett, in reviewing the chief measures which had been carried into effect during the past five years of his administration, referred to the travelling dispensaries in the following words :—

"About this time last year a suggestion was made by the Hon'ble Colonel Manifold which contained the germs of actual utility and enormous potentialities of future benefit. This was the suggestion to create travelling dispensaries as a plague-fighting agency. It was not expected that at their first inception these dispensaries would encourage an attitude of mind which would accept inoculation, evacuation and such principles of sanitation and hygiene as were likely to afford an environment unsuitable for the spread of the disease. These results will eventually follow, it is hoped; but they cannot be attained at the outset. But the measure stood out apart from all other measures so far suggested by the fact that it was economical, and that, even if it did not prove to be an effective engine against plague, it possessed other elements of usefulness which might justify it. As a result, 32 of these dispensaries were opened last year. They have been most successful and their number is being increased. It may be asked why, if the travelling dispensary is so useful, more money has not been spent on extending the experiment? The answer is, that this remedy, like its predecessors, has first to justify itself, and that to start it on a higher scale would have been more inadvisable. Efficient supervision was absolutely necessary if the dispensaries were to reach the people, and had the scheme been commenced on too ambitious lines, supervision would have failed. I do not think that one more travelling dispensary could have been added with advantage in the first year. Next year the number will be increased by 11 and such progress is quite sufficient for the second year of their work."

These experimental malarial travelling dispensaries proved so successful and popular that in 1911 it was decided to extend their number, a sum of Rs. 12,000 being budgetted to meet the cost of running 11 such dispensaries for a period of six months from 1st July 1911.

These were allocated to 11 districts, each being, as before, under the direct control of the Civil Surgeon, the only modification introduced being that they were all placed under the general supervision of the Chief Plague Officer, who, in turn, worked under the orders of the Inspector-General of Civil Hospitals.

In May 1911 the Government of the United Provinces obtained the sanction of the Government of India to the entertainment of 32 additional travelling dispensaries to be employed with a view to combating plague, the cost of their upkeep to be met out of the annual provincial plague budget.

At the same time the services of 4 commissioned officers of the Indian Medical Service were placed at the disposal of this Government for plague duty, mainly for the purpose of supervising the working of these 32 plague travelling dispensaries.

Thus during the cold weather 1911-12, 32 plague travelling dispensaries under 4 special charge officers and 11 malarial travelling dispensaries each under a Civil Surgeon were working (during this period three additional travelling dispensaries financed by District Boards were being run on exactly the same lines).

In May 1912 the Government of India, on the strong recommendation of the local Government sanctioned an increase in the number of plague travelling dispensaries to 42, with a fifth Indian Medical Service officer, the number of malarial travelling dispensaries at the same time being increased to 13.

At the present time therefore the total number of travelling dispensaries working in 33 districts in the United Provinces is 57.

I do not propose to advert in this paper to the great success which has followed the inception of these travelling dispensaries as enough has, I think, been said on this subject in the annual reports and Government resolutions of the past two years to make such unnecessary.

Numerous requests for information regarding the personnel, administration, equipment, and cost of travelling dispensaries having reached me, I propose to describe as clearly and succinctly as possible the exact manner in which our travelling dispensaries are administered and equipped.

Three types of travelling dispensaries, plague, malarial, and district board, have been mentioned above and these names, unless their object is explained, may possibly lead to confusion.

The equipment of all travelling dispensaries is exactly the same, they are all run on similar lines, the only reason it is essential to differentiate in nomenclature being for budget purposes, all three types coming under different budget heads, thus, all expenses in connection with plague travelling dispensaries are debited to the plague budget, the cost of the upkeep of the malarial travelling dispensaries is debited to a special varying grant sanctioned annually by the local Government for this purpose, and district board travelling dispensaries are paid for entirely by district boards who have voluntarily asked to have them placed in their districts offering to meet the cost of upkeep themselves.

As regards the present allocation of travelling dispensaries throughout the province a map shows the whole scheme at a glance.

The areas shaded blue contain the 42 plague travelling dispensaries and represent five circles, each containing 8 or 9 travelling dispensaries spread over four districts, and supervised by an Indian Medical Service plague officer.

All the other travelling dispensaries, *viz.*, 13 malarial (shaded red in map), and two district board (outlined yellow), are under the direct supervision of the Civil Surgeons of the 15 districts in which they work.

The Chief Plague Officer under the orders of the Inspector-General of Civil Hospitals, United Provinces, is responsible for the running of all travelling dispensaries.

By frequent touring he keeps in touch with Civil Surgeons, and Supervising Medical Officers, acquainting himself with the requirements of various districts, working out the lines on which the dispensaries should work, modifying these in accordance with the advice and wishes of the district officers, and the peculiarities and wants of particular districts.

He is also responsible for the maintenance of all stores and equipment, and exercises a general control over all expenditure.

The Indian Medical Service officers in charge of circles are practically always on tour, only returning to head-quarters periodically to replenish their own and the travelling dispensary stock of drugs.

All travelling dispensaries are in the charge of specially selected Sub-Assistant Surgeons, these being men of under 10 years' service.

It is advisable that only comparatively young active men should be placed on this duty as the life is a very hard and strenuous one, not suitable to the more senior men who frequently become less active in their habits owing to sedentary lives, and who would be discontented with duty which necessitates their continual absence from their families.

A considerable amount of the Supervising Medical Officer's time is taken up in inspecting the travelling dispensaries alone; each circle comprising as it does four districts, necessitates a good deal of travelling, much of it by train.

Each travelling dispensary is inspected by the Supervising Medical Officer, when possible at least once in every six weeks, in the case of plague dispensaries, more frequently.

During the season plague is prevalent Supervising Medical Officers spend as much of their time as they can conveniently spare inoculating against plague.

The field of surgical operation work carried out by travelling dispensaries is gradually spreading, and last year 446 major and 9,018 minor operations were done.

The major surgery performed by the Sub-Assistant Surgeons themselves consists only of such operations as require little or no after treatment such as fractures, dislocations, excision of cysts, etc.

The procedure we are at present adopting with regard to such operations as cataract, hydrocele, etc., which require careful after treatment is as follows :— the Sub-Assistant Surgeon first advises the patient to go to the Sadr (District Head-quarters) Hospital to be operated on by the Civil Surgeon, and, if he consents, he is given a special form of blue ticket to bring to the Civil Surgeon (See Appendix 1).

Unfortunately although this sounds an excellent plan in theory very few of these patients ever arrive at the Sadr Hospital, probably not more than about two per cent.

We are now by means of these blue tickets keeping a check on the number of operation cases which actually do come into the Sadr Hospitals, but even though the cost of conveyance is offered to poor patients the percentage who arrive is lamentably small.

Having tried in vain to get the thousands of operable patients our travelling dispensaries come across in the villages to go to the Sadr Hospitals, we now adopt the following procedure :—the Sub-Assistant Surgeon having arranged a date with his supervising medical officer collects such cases as wish for operation at some convenient centre, either in a house lent for the purpose, or, if such is not available, in *chappars* specially built.

The Supervising Medical Officer operates on these cases, stays a day or two afterwards in camp at the place, and then moves on, leaving the Sub-Assistant Surgeon in charge of the patients as long as is necessary for the after treatment, returning himself to dress important cases when necessary.

As I believe there is a mistaken idea that we operate on cases, and after operation allow them to look after themselves not arranging for their after treatment, I have specially brought out this point.

Other facts I would like to mention with regard to the operating work done by travelling dispensaries are—

(1) That our cases are in the first place advised to go to be operated on by the Civil Surgeon in the Sadr Hospital, being often offered conveyance allowance as an inducement ; the great majority however refuse, and these if not operated on at, or in the immediate neighbourhood of their own villages, often continue to suffer, die, or, in the case of cataract, fall into the hands of the *Sattiah* or "Lens coucher," of whom, notwithstanding all our district dispensaries, a goodly number still manage to exist.

(2) The Sub-Assistant Surgeons in charge of travelling dispensaries are not themselves allowed to do cataract operations, these being performed by their Supervising Medical Officers who are officers of the Indian Medical Service. (One Sub-Assistant Surgeon has received special permission, but he happens to be a man who is certified as thoroughly understanding the operation.)

(3) That the majority of the cases we meet would, if not operated on by travelling dispensaries, never go to the Sadr dispensary.

I will now describe the actual manner in which these travelling dispensaries work. Civil Surgeons or Supervising Medical Officers in consultation with district magistrates draw up an itinerary for each travelling dispensary of from a month to six weeks' duration.

Only the larger villages are selected to halt at, the travelling dispensary remaining for a period of from 5 to 15 days at each place, the Sub-Assistant Surgeon daily visiting the smaller villages within a radius of three to four miles. He submits a weekly report on a special form (see Appendix 2) showing all diseases treated by him during the week, the number of villages visited, plague inoculations performed, description of major operations, number of wells permanganated, etc. This weekly report is submitted to his Supervising Medical Officer, or, if he is working with a malarial dispensary, to the Civil Surgeon. They are forwarded by these officers to the Chief Plague Officer accompanied, in the case of the Supervising Medical Officers, with a diary (see Appendix 3) showing their own movements, inspections, plague inoculations done, plague cases treated, operations performed, in short anything of interest such as all facts ascertained regarding the epidemiology and endemiology of plague, cholera outbreaks, and all epidemic diseases. These weekly diaries and reports are forwarded, with any remarks he thinks necessary, by the Chief Plague Officer to the Inspector-General of Civil Hospitals. Monthly reports of the work done by each travelling dispensary are also submitted on a special form (see Appendix 4).

A traced district map showing the exact movements of the travelling dispensaries is also submitted monthly to the Chief Plague Officer and by him to the Inspector-General, which, after perusal, is returned to the Supervising Medical Officers to be used again.

A copy or précis of the weekly diaries is always sent by each supervising medical officer to the District Magistrates and Civil Surgeons of all districts in which travelling dispensaries work so as to keep them informed as to the exact part of the district the travelling dispensary is working in at any time. This is most advisable particularly when cholera is prevalent, as the Civil Surgeon can always at once move the travelling dispensaries to any villages in which cholera has broken out, provided he thinks this advisable.

We have in these dispensaries also a ready-made form of hospital which is always ready for immediate use in famine relief camps.

No travelling dispensary ordinarily works anywhere within a radius of 5 miles from any district dispensary to avoid covering the same ground and thus defeating one of the main objects of their existence, namely, the bringing of medical relief to the outlying tracts which are not supplied with fixed dispensaries.

The great success of these travelling dispensaries is beyond cavil and I feel convinced that their extension and development will well repay the expenditure involved.

The Sub-Assistant Surgeons have been granted a very liberal scale of allowances, but no horse allowance has up to this been given them as they rarely move more than 5 or 6 miles. Many of them, however, have purchased ponies for themselves and we have now asked Government to sanction a horse allowance of Rs. 15 per mensem to such Sub-Assistant Surgeons as are certified as possessing a pony—their own property. Although supplied with a tent, the Sub-Assistant Surgeon is frequently entertained in the villages and, in cases where he has no offer of hospitality, he can usually occupy the village *dharmsala* or school house; it is however essential that he be supplied with a tent for use in time of plague to avoid any risk of his contracting the disease.

One thing I consider absolutely essential to the successful working of travelling dispensaries is that they should be under good supervision and be inspected fairly often, otherwise I would not care to answer for the work reported as done. I know from experience that the advice and guidance of a medical officer is frequently needed by the Sub-Assistant Surgeons who, being quite junior men, often fail to show the most rudimentary ideas of discipline at first, they improve however, when working under the supervision of Indian Medical Service officers, out of all recognition in a very short time.

The following table shows at a glance the number of patients suffering from all kinds of diseases treated by travelling dispensaries during the year ending 30th June 1912.

TABLE.

Showing the diseases of the out-door patients treated in the travelling dispensaries in the United Provinces during the year ending the 30th June 1912.

Plague Inoculations.	INFECTIVE DISEASES.												
	Cholera.	Dysentery.	Enteric fever.	Gonorrhœa.	Worms.	Leprosy.	Malaria.	Plague.	Pneumonia.	Pyrexia of uncertain origin.	Rheumatic fever and rheumatism.	Small-pox.	Syphilis (primary and secondary).
1	2	3	4	5	6	7	8	9	10	11	12	13	14
16,293	1,155	14,603	19	2,184	27	613	77,733	2,165	119	389	22,908	77	3,126

INFECTIVE DISEASES—*contd.*		OTHER GENERAL DISEASES.						SYSTEMIC DISEASES.				
					New growth.							
Tubercle of the lung.	Other tubercular diseases.	Anæmia.	Diabetes.	Scurvy.	Non-malignant.	Maligant.	All other general diseases.	Diseases of the nervous system.	Diseases of the eye.	Diseases of the ear.	Diseases of the nose.	Diseases of the circulatory system.
15	16	17	18	19	20	21	22	23	24	25	26	27
683	549	7,023	65	14	194	118	6,791	11,119	73,167	14,438	1,825	530

SYSTEMIC DISEASES—*contd.*

All diseases of the respiratory system except pneumonia and tubercle of the lungs.	Dyspepsia.	Diarrhœa.	Abscess of the liver.	All other diseases of the liver.	Appendicitis.	All other diseases of the digestive system.	Inflammation and suppuration of lymph glands.	Goitre.	Bright's disease.	All other diseases of the urinary system.	Hydrocele.	Other diseases of the generative system.
28	29	30	31	32	33	34	35	36	37	38	39	40
49,893	20,539	7,506	12	1,257	280	30,495	2,004	758	173	1,361	462	2,761

SYSTEMIC DISEASES—*concld.*					GENERAL AND LOCAL.			OPERATIONS.	
						Poisoning.			
Disease of the organs of locomotion.	Diseases of the connective tissue.	Ulcers.	Other diseases of the skin.	All other local diseases.	Injuries (general and local).	By other poisons.	Total number of out-door patients.	Major.	Minor.
41	42	43	44	45	46	47	48	49	50
1,922	9,676	22,341	1,05,262	888	4,546	310	5,04,077	446	9,018

Surpervising Medical Officers, in addition to having all the instruments and English drugs supplied to the Sub-Assistant Surgeons in charge of travelling dispensaries, are given the following epuipment.

A liberal scale of tentage, as these officers have to be continually on tour, is most advisable, and the following have been supplied :—

1 Swiss cottage tent, 14′ × 14′, price complete Rs. 605.

1 Swiss cottage tent, 12′ × 12′, price complete Rs. 524.

These tents are of the special pattern supplied to the United Provinces police and are obtained from the Elgin Mills Company, Cawnpore.

I consider however that two 12′ × 12′ tents would be a more satisfactory scale as they are quite large enough and are easier to pitch and cost less to transport.

Four servants' shouldaries are also given to each medical officer and one shouldari for his clerk ; 2 camp tables and 2 camp chairs.

Each medical officer is supplied with a pair of panniers (A and B obtainable from the Medical Stores, Lahore Cantonment), these being of special pattern invented by Lieutenant-Colonel O'Gorman, I. M. S. (see photo. No. 1).

These panniers are excellent, being both light and strong, they are fitted with special containers for drugs and compartments for dressings, instruments, etc., their price per pair being Rs. 72.

While the greatest care has been exercised in selecting and limiting the variety of drugs and instruments supplied to the Sub-Assistant Surgeons in charge of travelling dispensaries, medical officers are supplied with practically anything in the form of drugs they require, except such as are prohibitive in price. It is very necessary to limit strictly the variety of drugs supplied to travelling dispensaries as the Sub-Assistant Surgeons continually ask for unnecessary drugs, many of which have similar therapeutical action to drugs already supplied ; as an instance of this I may mention that one man particularly asked that he be supplied with aspirin, phenacetin, antipyrin, and antifebrine.

The following instruments are supplied to each medical officer.

A special pattern pocket instrument case obtainable from Messrs. Peake Allen & Co., Lucknow, containing scalpel, bistoury, dissecting forceps, surgical scissors, clinical thermometer, 2 Spencer Wells' forceps, director, soft metal probe, needles and ligatures, price complete Rs. 22.

As these officers have splendid opportunities for doing a considerable amount of eye surgery, a five guinea set of eye instruments has been supplied to each (Weiss's instrument catalogue, No. 4456) ; these are obtained from the Medical Stores, Madras.

A central godown has been opened in Lucknow in order to enable us to stock and supply at once urgent replacements and requirements for travelling dispensaries such as urgent requisitions for all drugs, special tabloids and pills not stocked by Medical Stores which travelling dispensaries are supplied with, all spare parts for plague inoculation outfit, plague vaccine, stationery, special plague pills, etc.

Full details of the complete equipment supplied to each travelling dispensary are given in Appendix 6, I therefore propose here only to discuss briefly practical points with regard to certain articles which we have found useful.

As regards tents for the use of the Sub-Assistant Surgeons, I consider that the best scale would be to supply each man with one living tent for himself, the tent recommended being the officer's 80-lb. tent (field service pattern) obtainable from the Elgin Mills, Cawnpore, price Rs. 120 each.

One servant's pal 10′ × 8′ is required for his khalasi and servant.

When our travelling dispensaries were started I worked out a specially designed box and haversack suitable for carrying our drugs in pill, tablet, and

ointment form (see photos. II, III and IV). These have not been improved upon and have proved to be quite suitable in every way.

The first thing to be decided on was, were we going to have wet drugs in any form or should we limit ourselves entirely to pills, tablets, and ointments ? We decided on the latter as more suitable, being lighter and more compact, thus ensuring greater facility of transport.

This I consider a most important point as in the rains many parts of this province are quite impossible for bullock carts to get over, and if the equipment required bullock carts for transport, our travelling dispensaries would often be unable to move, as it is, the whole dispensary can always be carried on the heads of a few coolies. Again if bullock carts were always necessary during transit, the Sub-Assistant Surgeons would undoubtedly often have great difficulty in obtaining such, particularly during sowing and harvesting times.

I thought that the actual cost of the drugs we required, in pill and tablet form, would be roughly from 20 per cent. to 25 per cent. more expensive than wet drugs but considered that we would save at least this amount on transport, waste due to frequent bottle breakages, etc., also the fact that compounders are unnecessary as all dispensing is thereby done away with.

Of course no budget would stand the purchase of tabloids, etc., at the retail prices they are sold by small local chemists, so we asked the big manufacturing chemists to quote for large quantities of any drugs we required which were not stocked by the Medical Store Depôts. The quotations received were remarkedly low, and I found that we could actually adopt this dry system of drugs throughout, probably at a lower cost than if we used the more cumbersome wet drug system. When I mention that we obtain several of the drugs we use in tablet form at a rate of less than Rs. 2 per 1,000 this will be understood.

The retail rates charged on small quantities of these drugs appear to be out of all comparison with the manufacturers' cost of outturn. A retail chemist charges Re. 1 for a bottle containing 25 aspirin tabloids, we get exactly the same article in the form of Aceto Salicylic Acid made for our travelling dispensaries by the Medical Store Depôt, Madras, in tablet form at about Rs. 4 per 1,000 or 40 times cheaper.

Travelling Dispensaries ordinarily carry 25 varieties of drugs in pill, tablet or ointment form (*viae* Appendix VI).

The medicine box we supply is shown in photo. No. II, and is a plain wooden box 2 feet long, $1\frac{1}{3}$ feet wide and $1\frac{1}{4}$ feet high, which can be made by any *mistri* at a cost of about five rupees.

Originally we used empty Cadbury's chocolate tins as drug containers, purchasing these from the native bazaars in Lucknow, Cawnpore and Meerut. Having very soon exhausted the available supply we had similar tins, but enamalled with Ripolin inside and out, and of two kinds, cylindrical and rectangular, made by J. Johnson & Co., Aligarh. (See foreground of photo. VI.)

Enamelling the inside of the tins does away with the possibility of any chemical action taking place between the tin and such a drug as tablet Hydrarg Perchloride. The special type of lid supplied with Cadbury's chocolate tins is of advantage as being both air-tight and dust-poof.

This wooden medicine box contains, as shown by photo. No. II, a lift-out tray divided by partitions into 16 compartments, each to contain one tin. Under this tray the main body of the box is used for carrying ointments, dressings, etc., one side being divided up so as to carry 3 quart bottles, one containing rum (a useful stimulant for plague cases), a second castor oil, the third carbolic acid.

We now propose adopting an improved form of medicine box built on the same pattern for us by Messrs. Allibhoy Valleji of Multan and supplied at Rs. 38 each.

This box is strongly made of varnished wood bound with iron bands, the lift-out tray being made of tin, and having a space for carrying instruments at one

side, the compartments for holding the 3 quart bottles also lift out and are made of tin. (See photos. 5 and 6; also diagrams on plate VII.) A second ordinary wooden box of the same dimensions is also supplied to carry stationery, etc.

Each Sub-Assistant Surgeon is given a small leather handbag to carry his daily supply of dressings when he visits outlying villages (see photo. No. II); these we obtain from the North-West Tannery Company, Cawnpore, at a cost of Rs. 10 each.

The details of the special type of haversack we use are so well shown in photos. III and IV and in diagrams on plate VIII, that any further description is unnecessary except to state that there is a compartment at the base of the central partition to carry the pocket instrument case supplied, also a third row of spaces for bottles along the base of the posterior aspect of the central partition. This haversack is obtained from the Elgin Mills Company, Cawnpore, price Rs. 8 each.

A khalasi carries the haversack and handbag when the Sub-Assistant Surgeon makes his daily visits to outlying villages.

In addition to the above each man is supplied with a complete plague inoculation outfit.

The life of the Sub-Assistant Surgeons on this duty is a particularly hard and strenuous one, in addition to which there is the continual absence from their families.

Endeavouring to make things easier we are now experimenting with a form of caravan, details of which are shown in diagrams on plate IX.

This caravan, or "*Karanchi*" is strongly, though lightly, made and can easily be drawn by a pair of bullocks. The roof is surrounded with iron rails to enable the boxes and personal luggage of the Sub-Assistant Surgeon to be roped on. At the sides are *jhilmils* which can be opened at will. One each side are spacious lockers, which when shut, form seats. A double hinged seat is supplied to one of these lockers which, when let down at night, completely closes in the well of the *Karanchi*, thus forming a flush surface of the whole inside, on which if necessary, owing to the prevalence of plague or cholera, the Sub-Assistant Surgeon can sleep.

A seat also runs across the front of the inside of the *Karanchi* where a space has been left on which the medicine boxes can be carried. Above the entrance door at the back a canvas sheet is rolled up which, when opened out and supported by a couple of bamboo poles, forms a kind of verandah under which the Sub-Assistant Surgeon can work.

The cost of this *Karanchi* will probably work out at about Rs. 150 each, but if the two experimental ones we propose starting with, prove successful, they will enable us to do away with the necessity for any tentage for the Sub-Assistant Surgeons.

The cost of transit will also be very small as this will only mean a pair of bullocks for a short stage 3 or 4 times a month.

Hundreds of thousands of pamphlets and leaflets, on various diseases carefully selected and written in the simplest manner, have been issued through the medium of travelling dispensaries.

Copies of the following are attached :—

Leaflets on plague, its cause and prevention (Appendix 7).

Plague pamphlets (illustrated) (Appendix 8).

Leaflet advocating evacuation (Appendix 9).

Leaflet on malaria (Appendix 10).
Illustrated posters on malaria (Appendix 11).
Leaflets on cholera (Appendix 12).
Leaflets on small-pox (Appendix 13).
Leaflets on consumption (Appendix 14).

As it was found that the villagers, when given a white tablet, were dissatisfied, thinking all such to be quinine, we now supply these in various colours, blue, green, yellow, etc.

We have discarded tablet Blaud Co. for a more stable tonic tabloid which Messrs. Burroughs and Wellcome have supplied to our own formula.

R/

Ferri et ammonii citratis grs. III.
Quinine sulphatis gr. I.
Extract cascara } a. a. gr. ½.
Extract gentian }

A special stimulant pill for plague is also supplied consisting of—

R/

Ammonii carbonas grs. 3½.
Pulv Digitalis gr. 1.
(physiologically standardised)
Strychninæ, Phosphas gr. $\frac{1}{32}$.

All supervising medical officers have reported well on the results following the administration of the above pill, but owing to the formation of a sub-carbonate of ammonia, any but fresh stock is useless.

Most of our instruments and drugs we now obtain direct from the Medical Store Depôt, Madras, the greater part of the latter being manufactured there at a minimum cost.

This we have found most satisfactory, the drugs being invariably fresh and all orders very promptly executed.

The bazaar drugs shown in Appendix VI are also supplied and their use is being gradually extended wherever the travelling dispensaries have already established a reputation; these are periodically made up at Supervising Medical Officers' head-quarters by compounders.

Personnel and annual recurring cost of our travelling dispensaries.

I.—*Supervising Medical Officers.*

These officers average say 6 years' service and receive the consolidated rate of pay for Indian Medical Service officers on plague duty as laid down in Secretary of State's No. 60 (Revenue), dated the 8th May 1908—this for an officer of 5 to 7 years' service is Rs. 800 per mensem. Average travelling allowance Rs. 150 per mensem.

Staff of Supervising Medical Officers.

	Rs.	
One clerk on	25	per mensem.
Average travelling allowance of clerk under Civil Service Regulations	=15	„ „
Two peons on	7	„ „
Average travelling allowance	=5	„ „
Two khalasis on	6	„ „
Average travelling allowance	=5	„ „

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II.—*Subordinate Staff.*

Monthly pay and allowances of Sub-Assistant Surgeon :—

	Rs.	
Pay of 4th grade Sub-Assistant Surgeon	30	
Local allowance	25	
Travelling allowance under Civil Service Regulations average	15	
Total ...	70	
Khalasi for Sub-Assistant Surgeon on	6	per mensem.
Average travelling allowance	3	

The cost per annum of running one travelling dispensary—

	Rs.
Pay and allowances of Sub-Assistant Surgeon as detailed above at Rs. 70 per mensem	840
Pay and travelling allowance of khalasi	108
Cost of medicines excluding quinine at Rs. 60 per mensem	720
Carriage of medicines, tentage, etc., average at Rs. 20 per mensem	240
Contingent and miscellaneous charges	120
Total ...	2,018

or say Rs. 2,100 per annum for each travelling dispensary.

To this should be added the cost of quinine (tablets grs. *III*) at Rs. 10 per lb.—about Rs. 300 per annum.

The cost of quinine must necessarily be a variable one as the quantity used, depending entirely as it does on the prevalance or otherwise of malaria in epidemic form, cannot be forecasted.

In the event of malaria existing in epidemic form the cost of quinine over and above the quantity mentioned has to be borne by district boards ; the same applies in the case of permanganate of potash used in disinfecting wells in districts where cholera exists in epidemic form.

Our travelling dispensaries are now in the third year of their existence and it is most satisfactory to note that, not only has there been no decline in their popularity, but as stated in the Government Resolution on the Hon'ble Colonel Manifold's plague report for 1911-12 "a particularly gratifying feature in the reports on their working is the abundant testimony to their popularity with all classes."

The District Boards of several districts have offered to meet the cost of maintaining a dispensary from their own funds, and following the departure of a dispensary, petitions have been received from the people asking for the opening of a permanent dispensary, the funds for which they have in some cases voluntarily subscribed. The Lieutenant-Governor is convinced that these dispensaries constitute a most important departure in medical administration, and that their future development and extension will well repay the expenditure involved.

ALL-INDIA SANITARY CONFERENCE MADRAS NOVEMBER—1912.

Anti-Malarial Measures—Itinerating Dispensaries.

By the Hon'ble Surgeon-General W. B. BANNERMAN, C.S.I., M.D., I.M.S.,

Surgeon-General with the Government of Madras.

This measure for the prevention of malaria has been tried in the Gōdāvarī district of the Madras Presidency for the past two years with, I fear, but a scant measure of success.

The operations were confined to two portions of the Chodaveram division of the Gōdāvari district, which were mapped out and systematically visited by two Sub-Assistant-Surgeons each working independently with a travelling dispensary. The villages in each portion were to be visited every two months. Those suffering from fever to be treated with quinine, and enough of the drug left behind for prophylactic use during the absence of the dispensary.

The work resolved itself into the administration of quinine, and the putting forward of recommendations for the filling in of hollows.

No attempt was made to explain to the people in a simple and interesting manner the cause of malaria, nor was instruction in its prevention given.

Probably the Sub-Assistant Surgeons were not in a position to do this, for they were not provided with apparatus for demonstration nor with diagrams or lanterns and slides for this purpose. It is clear that they did not appreciate the matter of prevention themselves, for they both became so ill that the tours, for this reason, ended on 31st December 1910, having lasted only four months instead of six, as originally planned.

The District Medical and Sanitary Officer under whom the work was carried out reports that the quinine was mainly appreciated by plain dwellers temporarily residing in the Hill Tracts, rather than by the hill people for whom it was intended. It was likewise found that quinine left behind in villages was not much used by the people when free of fever, and in some instances was thrown away after the departure of the dispensary.

The District Medical and Sanitary Officer believes that the small success of the trial was due to the ill-health of the Sub-Assistant Surgeons, to the difficulty of travelling in a wild and hilly country devoid of roads, and to the smallness of the villages.

The results obtained were very disappointing. The number of villages visited during the four months was 141, and the splenic index reported was 39·2 per cent. Yet the numbers allowing themselves to be treated were disappointingly small. The actual totals were—

	470	treated for malaria.
	728	,, ,, spleen.
	2,226	,, prophylactically.
Grand total ...	3,424	

It therefore appears that in each village only—

3·3	persons	were	treated	for malaria.
5·1	,,	,,	,,	,, spleen.
15·7	,,	,,	,,	prophylactically.

In the following year the dispensaries worked in the same part of the district from September 1911 to the end of January 1912. The total number of villages visited was 136 and the number of patients treated for fever 107. Those treated prophylactically numbered 5,767.

It will be seen that the results are only slightly better than in the previous year. The number of fever cases treated in each village averaged less than one person (actual 0·78) and is even fewer than in the previous year.

The number of persons treated prophylactically is however higher, being 42·4 for each village. This seems a disappointing result of the work of two men for five months; working out, as it dees, at about twenty-three cases each a day, taking twenty-five working days in the month.

With this practical experience before us, the Sanitary Commissioner and I have drawn up a scheme on the following lines and submitted it to Government for sanction and we should be obliged if delegates who have had greater experience than ourselves would discuss this important matter with a view to perfect the design.

We believe that these travelling dispensaries should be much more than mere drug dispensing concerns. They ought to be used to diffuse knowledge among the villagers with regard to the cause and prevention of malaria.

For this purpose the Sub-Assistant Surgeons appointed should be carefully trained and selected. They should go through a course of instruction under the Malaria Expert, in the life history of the mosquito and the malaria parasite. They should be taught to distinguish between the malaria—bearing and the harmless mosquitoes. They should be drilled in the life history of the malaria parasite in man and in the mosquito. They should be shown these things and *made to believe in them*, so that they may become enthusiastic in convincing others. They should likewise receive instruction in taking the splenic index, and in making a census of fever cases.

In the matter of prevention they should be taught the proper use of the mosquito net, and how to give quinine in the best manner.

They must be given a lantern and slides, and attractive diagrams, and instructed in the art of the popular lecturer. This would enable them to give lantern lectures in the evening at the village chavadi.

Lastly they should be provided with nets and other apparatus for catching larvæ to enable them to demonstrate to the villagers that mosquitoes *really* do originate from the "wrigglers" in the pools. This latter plan was tried with considerable success on the lands of the Improvement Trust in Bombay by Mr. Orr, the Chairman of that body.

PART II.

PLAGUE.

ALL-INDIA SANITARY CONFERENCE, MADRAS—NOVEMBER 1912.

The Annual reappearance of Plague.

LIEUTENANT-COLONEL S. BROWNING-SMITH, I.M.S., CHIEF PLAGUE MEDICAL OFFICER, PUNJAB.

DEFINITION.

In the Punjab the annual epidemic of plague is at its height in March, April and May, and during the last mentioned month, with the advent of hot-weather conditions, rapidly declines until by the middle of August very few, if any, signs of active infection remain; a little later an increase shows the beginning of the next visitation. From the experience gained from watching the onset of several epidemics and from facts gathered from a careful and detailed investigation of every infection in the latter half of 1908, a particularly favourable time for accurate inquiry, I attempted to show in a paper read before the Bombay Medical Congress, 1910, that the annual visitation in the Punjab had its principal origins, not in the few places where plague lingered on into the summer, which as a rule were of minor importance, but in numbers of places where active signs of infection, rat mortality and human plague, reappeared after a longer or shorter interval during which no such signs were apparent and where no fresh importation of plague had taken place. A summary of this paper is given in the appendix. In part I of this paper it is proposed to make a critical examination of the facts in connection with the appearance of plague in the autumn of 1911, and in part II to discuss generally this phenomenon in the light gained by the present and past investigation.

PART I.

The present inquiry.

During the latter half of the year 1911, owing to the partial failure and late arrival of the monsoon, the autumnal increase in plague was much less than usual, and, with only a comparatively small number of localities showing infection, it was possible to investigate all plague appearances between the 1st August and the 15th December in detail with the object of determining to what extent those places showing plague infection persisting throughout the summer were responsible for the subsequent epidemic. A particular interest attaches to this inquiry from the unusual occurrence of a considerable epidemic, which was in active progress throughout the summer in a large town, Rawalpindi, on the main line of railway. The cases and deaths from plague recorded every week in each district and native state in the province are tabulated in Statement I attached, and it will be clearly seen that in the majority of districts the gaps of freedom are considerable and extend to months in the case of southern districts such as Delhi, Hissar, Rohtak and Gurgaon, where hot weather conditions are established earlier and are more prolonged than in the north; these figures are those notified weekly by the official reporting agency, but corrected, as far as possible, by the investigations of the plague medical staff.

Persistence of plague throughout the summer of 1911, and diffusion of infection therefrom (Statement II and map A.)

Places which showed actual signs of the presence of infection practically continuously throughout the summer were only three in number, (see Statement II (*a*), Rawalpindi City, and two villages, Narot in the Gurdaspur district and Mallupota in the Jullundur district.

(1) *Rawalpindi City* and the adjoining cantonment. The district was infected in the spring and five cases were imported into the city, three from the district, one from Lyallpur and one from Gujrat in the latter half of April; the

first indigenous case occurred on the 6th May, in connection probably with the Gujrat infection; from that time the epidemic continued throughout the summer and winter, and 596 cases with 393 deaths were reported up to the 15th December; Rawalpindi, therefore, constituted an active plague centre throughout the quiescent period and although in the hot months cases were few, in only one week was there no report of plague (see Statement I).

(2) *Mallupota, Jullundur district.*—A village of 1,027 inhabitants, four cases and three deaths occurred here between 4th April, and 22nd June, rat mortality was noticed at intervals during the hot weather and on November 17th a human case occurred, and sixteen further cases with 7 deaths between this date and the 15th December. Although the history of rat mortality is indefinite, this may be taken to be one of those cases where persistence of infection throughout the quiescent period is marked by occasional observable rat mortality. This type is comparatively uncommon compared with those places where persistence is not so marked, but is a link between the continuous epidemic type, Rawalpindi, and those cases where there is a gap of apparent freedom.

(3) *Narot, Gurdaspur district.*—A village of 844 inhabitants; an epidemic occurred here in April and May, rat mortality is said to have been noticed in June, July and August, and again in October, the first human case occurred on 5th November, only six cases in all with four deaths; there was no extension from this centre. In this instance there was a period of some weeks when no rat mortality was observed.

The above were the only three places in the Punjab where the persistence of infection, *as evidence by the recognition of rat mortality and human plague or both*, was located; the inquiry will now be directed to ascertaining to what extent these centres led to the annual epidemic in the province by diffusion of infection through importation.

Importation from these persistent centres.—Of the three centres, Narot may be at once dismissed, the autumn epidemic was very slight and no spread of infection took place from it to other localities; the Mallupota epidemic only led to the infection of one village, Kaleran, about a mile distant from it; there remains then only the town of Rawalpindi in the extreme north of the province.

Up to the 15th December, forty-eight cases of plague are known to have been exported from Rawalpindi to other parts of the province. It is of course known that a person may carry infection from one place to another without actually suffering from or incubating the disease at the time, but it is also well recognised that these cases are in the minority, and in the majority of instances the carrier himself is attacked; actual imported cases of plague may therefore be taken as a fairly accurate index of the extent and direction of the diffusion of plague from an infected centre. In this respect map A shows very typically what generally occurs. In the first place there is diffusion into the surrounding country spreading out from the centre both by road and by rail, up to the river Jhelum, but beyond this river, diffusion by road is checked, and is then only marked along the lines of rail, gradually decreasing in intensity with the distance from Rawalpindi; of the 48 cases, 28 were imported into the surrounding country between the rivers Indus and Jhelum, including three to places on the railway south of the Jhelum river, 15 were to places on the line of rail, and the remaining five to places near the line. Where the centre is not on the railway, diffusion by rail is less marked and may be said to vary inversely with the distance of centre from the railway. Up to December 15th these forty-eight plague cases had led to indigenous cases and an epidemic in only two instances.

(1) Saman, a village in the Attock district, population 1,189. A pneumonic case imported from Rawalpindi on 1st November, led to a short and sharp outbreak entirely of pneumonic plague, eleven cases all of which were fatal. The epidemic was soon over and no places were infected from this centre.

(2) Khushab, a town on the railway and on the north bank of the river Jhelum infected on 25th November by an imported case of bubonic plague; three indigenous cases following this importation had occurred up to the 15th December.

One other infection is attributed to Rawalpindi, and in this case the carrier was not attacked by the disease, the facts are as follows :—

(3) Kadirabad, Gujrat district, population 3,119. Two persons travelled from Rawalpindi to Bahauddin by rail and from the latter place to Kadirabad by country cart ; the driver of this cart was attacked by plague on 8th December 1911, and other cases among his family followed ; up to 15th December ten cases, all fatal, had occurred ; infection had not spread to any other place.

By the 15th December 1911, as the result of persistence of infection throughout the summer as evidenced by human cases and rat mortality, there were therefore five epidemics in progress, firstly Rawalpindi and its two derivatives, Khushab and Kadirabad, secondly Mallupota and its extension, Kaleran.

But a reference to Statement I shows that at this time plague was being reported from eighteen districts of the province, extending to its southernmost limits, and that, in spite of an active epidemic in progress in Rawalpindi since May, and a considerable exportation of plague infection from this centre, the whole tract of country between the rivers Indus and Jhelum, containing the districts of Rawalpindi, Attock and Jhelum and part of Shahpur, remained free of infection except for one place, Khushab, where four cases of plague had occurred. (Map A).

The conclusion is therefore natural, indeed it is inevitable, that the provincial epidemic was not the result solely of these infections which were observed to persist through the summer, as a matter of fact, such infections had caused but a very small proportion of this epidemic, and the origins of the greater proportion must be looked for elsewhere ; they either lay in the importation of plague from without the province or in the appearance of infection within, with no importation of infection to account for it.

Infection from outside the province.—Ten cases of plague were imported into the province from outside ; one from Dehra Dun in the United Provinces to a village Jando Singha (near rail) in the Jullundur district, one from Bhilsa to Gujrat town (on rail), two from Bhopal to Hodal (on rail), two from Hyderabad to Narnaul (on rail), one from Hyderabad to Nawazpura (near rail); none of these seven cases led to any further infection. Three cases were imported from the Jammu State into villages on the adjoining border of the Sialkot district, leading to the infection of a group of six villages close together,—Balipur, Chak Binjra Gujran, Kundal Khalka, Kashinand, Dalliwali and Darya Biddar—and also of another village on the border, Khoji Chak, at some distance from this group. No other infection could be traced from these, they are included in Map B, and this map clearly shows that epidemic plague, caused by importation from outside the province, was limited to a small part of the extreme border of the province and up to 15th December was strictly localized and had not spread any distance into the interior.

Sporadic cases during the quiescent period.—A certain number of isolated cases of plague occurred during the hot weather in places which, except in one instance, had been apparently free from infection for some time ; a list of these is given in Statement III ; in one place there were two such cases, in the others only one ; in no instance was any further infection observed up to 15th December and no diffusion of infection was traced to them. Cases of this sort are not infrequent, a single plague case or slight rat mortality occurring in a place which has been apparently free for some time and indicating that infection still persists; these cases may be dismissed with the remark that in only one instance did they appear to have any connection with subsequent infections and in that solitary instance to only a single contact case, they are therefore unimportant; the interval between these cases and the previous epidemics was generally long, they indicate that infection is persisting in spite of the absence of apparent signs, but as they were not followed by epidemic plague in the autumn they may be taken to be the last flickerings of the spring epidemic. The sporadic cases are shown in Map B.

Autumnal reappearance 1911, (Statement V and Map B.)—The infections that have been discussed were, however, but a small proportion of the total number of localities affected by plague by the end of the year; from August

onwards plague was reported from a large number of places where investigation failed to discover any evidence of importation from active centres, not only definite evidence but also evidence of any connection whatever with other infected places; wherever connection has seemed remotely probable the cause of the epidemic has been ascribed to importation. The following is a brief summary of the course of events always excepting the epidemic in Rawalpindi City in the extreme north; by the end of August the only districts that continued to report epidemic plague, the ends of the previous seasonal epidemic, were Sialkot and Amritsar and by the middle of August these were at an end; with the exception of the few single sporadic cases which led to no further evidence of the disease and which have already been referred to, no district was affected with epidemic plague up to the end of September when the first reappearance was reported from the district of Gurdaspur, central Punjab; during October plague reappeared with no importation to account for it in the districts of Gurgaon, Montgomery, Gujranwala, Hissar, Rohtak and Karnal; by the end of November fifteen districts all over the Punjab were reporting indigenous cases; this almost simultaneous reappearance of epidemic plague, each infection slight in itself, all over the central and southern Punjab is a particularly significant fact which should be carefully noted, plague never spread in the Punjab in this way as a result of importation alone; between the 1st August and 15th December plague had reappeared in thirty-eight widely separate localities without any evidence of importation to account for it, and had spread by importation from these to eleven other localities; in all cases these reappearances were in villages distant from the railway line and in no case did such appearance occur in a town or village where there was a railway station. A list of these reappearances, with no importation to account for them is given in Statement V (*a*) and a list of places infected from these by importation in Statement V (*b*) and (*c*).

A comparison of maps A and B.—Map A shows all the infections in the province that could be traced to those places where active signs of plague persisted during the quiescent period; Map B, on the other hand shows the infections that could not be traced to this source, and includes sporadic cases, and infection imported from outside the province. The maps show the whole area of the Punjab plains which is annually affected by plague; the north-eastern border of these plains is formed by the line of the lower Himalayas, extending from Murree on the north, extending through Jammu State and forming the edges of the Sialkot, Gurdaspur, Hoshiarpur and Ambala districts: plague has not yet spread in these hills; that part of the plains not included on the left of the maps has been but very slightly affected. A comparison of these two maps shows in a most striking way some very important facts; it will be noticed on the one hand in Map A that the active centre for the exportation of plague infection, Rawalpindi City, is situated in the far north of the province and that such exportation follows perfectly typical natural lines, by road and rail into the surrounding country until the river Jhelum is reached beyond which it is limited to places on the line of railway or its neighbourhood. On the other hand Map B, the places where epidemic plague reappeared in the autumn, were principally in the central and southern parts of the province, and, moreover, such reappearances were generally in villages remote from the railway and from each other; this factor of remoteness would be much more apparent if the maps were drawn to a larger scale—it should be remembered that the scale is 1 inch to 32 miles. A glance at the two maps should be sufficient to dispose of the fallacy, a fallacy at any rate as regards the Punjab, that the annual epidemic is solely to be attributed to those places where rat mortality, that is to say observable rat mortality and human plague persists throughout the quiescent period and to the diffusion therefrom by importation. Were this true, at the end of 1911, epidemic plague should be principally in evidence in the Rawalpindi and neighbouring districts of Jhelum and Attock, and, south of the river Jhelum, in the more important places along the railway line with areas of diffusion round them, exactly the contrary to what actually occurred. A glance forward makes this still more evident, for at the end of March the epidemic in Rawalpindi City was flickering to its end and the whole area between the rivers Indus and Jhelum was free from plague except the small town of Khushab and two neighbouring villages, and the maximum of infection was in the central and southern districts.

The only arguments that can be raised against the truth of these epidemiological facts are either (*a*) that certain sources of infection where plague persisted through the summer had escaped observation, or (*b*) that some factor other than importation by the human agent was at work. Such arguments hardly need refutation, but it may be said that in the first case (*a*) it is not reasonable to suppose that while in the north a large number of infections were discovered (in the majority of instances single imported cases) and all were traced to their origin, in the south in a large number of cases, mostly epidemics, the source of infection was overlooked, the reporting and investigating agency being the same in both cases ; in the second case (*b*), presuming the importation of plague by some undiscovered factor, it would be necessary in order to fit the fact disclosed by the two maps, to admit that the better and closer the communication with the infecting centre the less this factor was operative.

The Delhi Area.

A full list of the places where signs of infection reappeared after an interval of apparent freedom, without any fresh importation to account for it, is given in Statement V (*a*). It is not intended in this inquiry to give a detailed account of each separate infection ; the salient facts are the same everywhere and it will be sufficient for the purpose if a close examination is made of a particular area ; the tract of country round Delhi is eminently suitable for the purpose. In view of the Coronation Durbar at Delhi, special operations were undertaken in the surrounding country during 1911 ; this tract of country containing a population of some one and a half millions, consisted of the three revenue sub-divisions of the Delhi district and the five contiguous revenue sub-divisions, parts of the Gurgaon, Rohtak and Karnal districts ; the special plague staff consisted of one Indian Medical Service officer, one Civil Assistant Surgeon, and one Sub-Assistant Surgeon with subordinate staff for each of these eight sub-divisions, also one Indian Medical Service officer for the Durbar area. With such a staff it was possible to keep this part of the country under very close observation and carefully watch the end of the 1910-11 epidemic, the quiescent period during the summer and the beginnings of the epidemic of 1911-12 ; the attention of the civil staff was specially directed to the importance of reporting at once all plague occurrences including suspicious human cases and rat mortality, moreover substantial rewards were freely offered to any one giving information. Judging from the reports received, even of single rats which had met fatal ends from meeting a cat or falling down a well, I believe that no plague occurrence can have escaped notice ; many cases were reported which on investigation were proved not to be plague, they were usually cases of secondary glandular enlargement following septic wounds. This area is shaded in Map B.

The following is a summary of the plague occurrences in this area during the quiescent period and up to December 15th 1911 :—

The last case from this area was reported at the end of June.

On 29th July, a beggar was brought unconscious to the general hospital in Delhi City and was removed to the plague hospital where he died ; it appeared that he did not belong to the town and it could not be discovered where he had come from ; although this case was reported as plague it is very doubtful if it was so.

On September 5th, a plague case was imported from Bhopal into Hodal, a town on the rail, just outside the area, and on September 24th another from Bhopal into the same town.

On October 17th, a case of plague was imported from Rawalpindi to Behora Kalan, a village in the Gurgaon district, the case and contacts were isolated and not allowed to enter the village.

On October 21st, a case of plague was imported into Delhi City, the man was from Rawalpindi but was convalescent having stopped for some days *en route* at Amritsar City.

On October 24th, a case of plague from Rawalpindi, moribund, was discovered at Jatauli, a railway station, Gurgaon district.

The above are the only cases of plague known to have occurred in this area between the end of June and the middle of October, they were all imported and none of them led to any further infection, they had no connection whatever with the following appearances of epidemic plague.

On the 14th October, the first case of an epidemic occurred at Khajirka, Gurgaon district; on the 26th October, an epidemic began at Narah, Karnal district; on the same date another started at Pakasma in the Rohtak district, which imported infection into the neighbouring village of Bhalot, half a mile distant, on November 20th; on the 2nd December an epidemic started in Bakarwala, Delhi district. These were the only five epidemics in this area up to 15th December, and in four of them no evidence whatever could be found of importation. A reference to the map shows that these four reappearances were widely separated from each other, each in fact was in a different district and no trace of any connection between them could be discovered or between them and the imported cases noted above, or between them and other infections in the province; none of them were on the railway line.

(1) *Khajirka*, a small village of 651 inhabitants, about four miles from the town of Palwal (rail). This village was not infected during the previous season, but the neighbouring large village of Alawalpur suffered a severe epidemic, the last death occurring on the 31st May 1911. Rat mortality was first noticed in Khajirka on the 1st October 1911 and the first case of plague appeared on the 14th of the same month; by the 15th December some forty deaths had occurred. No spread of infection took place from this centre, one case only being exported and that to the village Alawalpur on 20th November; no epidemic resulted from this case. There was absolutely no evidence of importation of infection from outside, and it must be remembered that the movements of the inhabitants of these small villages and the arrival of people, beggars, relatives, etc., from outside is very accurately known; moreover the only places in the province where plague was epidemic when rat mortality started in Khajirka at the beginning of October, were (*a*) Basso Kot and Shahpur Jagan, villages in the Gurdaspur district (not on rail) at least 300 miles distant, and (*b*) Rawalpindi City, some 500 miles to the north; it is quite impossible for infection to have been imported from these places without being traced.

The only explanation, therefore, for this reappearance of plague and almost certainly the true one, is that infection was imported in the spring from the neighbouring village of Alawalpur, and that, although this was marked by no rat mortality or plague cases (not even an imported one) the rats then became infected, but there were no signs of this infection throughout the summer, and it was not until the conditions for plague became favourable in the autumn that infection became manifest.

(2) *Narah*, a medium sized village, population 2,724, in the Panipat Tehsil of the Karnal district some distance from any railway station. This village suffered an incomplete epidemic in the spring, eighteen cases and twelve deaths occurring during May—first case May 2nd, last case May 29th. Nothing further happened till about the 22nd or 23rd October when dead rats were noticed and on the 26th October five cases of plague occurred, followed by three on the 27th, then nothing till a single case on November 20th and in December further cases. There was no infection anywhere near and no evidence whatever could be obtained of any possible source of importation to which the origin of this epidemic could be attributed. No spread took place from this centre.

(3) *Pakasma*, a village in the Rohtak district, some distance from rail, population 2,606. This village suffered from an incomplete epidemic lasting from the 1st of May to the 12th of June 1910; plague reappeared on the 9th October 1910; and the epidemic lasted till the 13th January 1911, subsequent to this however there were four suspicious deaths, recorded as due to "fever" of short duration, the last of these in March, 1911: nothing further occurred till the 26th October when an epidemic began, resulting in 89 cases with 46 deaths up to the 15th December; rat

mortality was not noticed before the first case, but rats died in enormous numbers during the epidemic. No evidence whatever of importation could be elicited. From this village the neighbouring village of Bhalot was infected on the 20th November.

(4) *Bakarwala*, a small village of 733 inhabitants, not on railway, about twelve miles from Delhi City. This village had never previously suffered from plague; six cases of plague were imported into this village in April 1911, all of them proving fatal; three of these occurred in one particular house; no rat mortality or human plague cases of indigenous origin followed in connection with these imported cases at the time, but on December 2nd a case of plague occurred in the identical house where the three cases had arrived in the spring; no evidence of importation could be elicited to account for this indigenous case, except the occurrence in the preceding spring; between the importation and the indigenous case was an interval of seven months during which there were no signs of infection; the village was under close observation as a potential danger and was frequently visited during the summer and autumn. This is one of those very interesting cases where indigenous plague reappears after a considerable interval *in the same place* as the previous localized infection.

Although the above were the only infections in the area under special observation, another reappearance may be cited here as it is the only other infection in the southern Punjab.

(5) *Umra*, a large village, population 2,506, some distance from rail in the Hissar district. This village was not infected in the previous spring, nor were any of the neighhouring villages, some rat mortality however was noticed in May 1911 unaccompanied by any human cases; nothing further was noticed till the 1st October when rat mortality reappeared, and the first case of the epidemic occurred on the 24th October; 68 cases and 40 deaths up to 15th December; there was no extension from this centre. But rat mortality in May marks the presence of infection at that time, source unknown; there were however many epidemics raging at this time though there were none very near, and importation probably took place then. No evidence whatever could be obtained of importation to account for the origin of the October infection.

This completes the list of epidemics in the southern Punjab; it will be noticed that four out of the five reappearances unconnected with fresh importation occurred very much at the same time, and in all the distance between them was very considerable; there was not the remotest connection between them, they were all far removed from any other infections in the province, there had been no human cases of plague in the area for some months except the five imported cases which in no instance had any connection with these epidemics. Taking all the facts into consideration the presumption that infection was imported from some unknown mythical source into these five places is absolutely untenable, especially when it is remembered that known exportation in the north of considerable extent only lead to secondary epidemics in a small minority of cases. These five epidemics are undoubtedly instances of the reappearance of plague as indicated by rat mortality and human cases after the lapse of a considerable period of time during which no such signs of the persistence of infections were manifest.

With regard to this area it may be also noted that slight rat mortality was observed in fourteen places between the 1st August and the 15th December, with no accompaniment of human cases; that there was no epidemic in these cases may perhaps be attributed to the vigorous rat destruction that was undertaken at once in all. No connection was traced between any of these places and the epidemics that have been described, they are all however instances of the reappearance of infection after an interval of apparent freedom.

Conclusion.

The collection of epidemiological facts associated with the quiescent period 1911 and the beginnings of the annual epidemic 1911-1912 have been recorded, and these facts are in complete concordance with past experience. Theoretically it would be expected that epidemic plague would remain in evidence continuously throughout the summer in the large towns and that with the advent of favourable

conditions for plague activity in the autumn, infection would spread therefrom by road into the surrounding country from village to village, and by rail to much greater distances to the most important railway towns which would also become diffusion centres for the spread of infection by road into the neighbouring country. Practically, this is not what appears to happen; in the present investigation it is true epidemic plague continued throughout the summer in an important railway town; this however is an unusual occurrence in the Punjab, but it has been shown that, in spite of this persistent epidemic, the great proportion of the beginnings of the 1911-1912 visitation lay, not in the diffusion of infection by importation from this active centre, but in the reappearance of plague in villages where no importation could be discovered to account for it; moreover these appearances were widespread over the province, generally in villages not near the rail, remote from each other as regards distance but close as regards time.

Dismissing the sporadic cases and epidemics due to importation from outside the province, the provincial autumnal infection up to 15th December 1911 can be divided into two distinct groups.

(*a*) The three persistent centres from which 50 importations were traced which led only to four secondary epidemics, total seven epidemics in all, see Statement II.

(*b*) Reappearance, unconnected with importation, in 38 places leading to eleven epidemics, secondary to these, total 49 epidemics, see Statement V.

STATEMENT I.—*Showing Plague cases in the Punjab from the 1st July 1911 to the 16th December 1911.*

(Imported cases only are marked in antique.)

No.	Districts.	8th July 1911.	15th July 1911.	22nd July 1911.	29th July 1911.	5th August 1911.	12th August 1911.	19th August 1911.	26th August 1911.	2nd September 1911.	9th September 1911.	16th September 1911.	23rd September 1911.	30th September 1911.	7th October 1911.	14th October 1911.	21st October 1911.	28th October 1911.	4th November 1911.	11th November 1911.	18th November 1911.	25th November 1911.	2nd December 1911.	9th December 1911.	16th December 1911.	Remarks.
1	Hissar	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	2	3	7	6	2	13	...	40	Spring last case, week ending 17th June.
2	Rohtak	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	6	7	9	3	7	20	10	Spring last case, week ending 24th June.
3	Gurgaon	...	...	...	...	...	...	...	...	...	1	...	...	1	...	1	3	2	3	16	9	8	4	1	...	Spring last case, week ending 17th June.
4	Delhi	...	...	...	1	...	...	...	...	...	...	...	...	...	...	1	...	...	...	...	...	...	...	2	6	Spring last case, week ending 17th June.
5	Karnal	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	8	...	...	18	...	21	7	14	Spring last case, week ending 1st July.
6	Ambala	...	...	...	...	1	2	1	...	...	...	1	...	...	...	...	...	...	...	3	5	8	17	12	25	Spring last case, week ending 17th June.
7	Simla	These districts seldom and very slightly infected.											...	...	...	...	...	...	...	...	...	...	...	...	...	
8	Kangra																									
9	Hoshiarpur	3	4	4	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	10	...	...	...	9	4	
10	Jullundur	...	3	3	1	...	1	...	...	...	...	...	...	...	...	...	...	...	1	...	...	2	3	7	3	
11	Ludhiana	...	...	...	...	...	...	...	...	...	...	1	...	...	...	...	...	...	...	5	5	7	14	5	3	
12	Ferozepore	..	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	2	...	
13	Lahore	152	74	35	4	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	...	10	
14	Amritsar	104	...	15	...	16	...	...	1	...	...	...	...	...	...	1	...	...	1	...	...	...	...	...	...	
15	Gurdaspur	464	34	19	1	...	1	...	...	...	...	...	1	8	...	4	...	...	...	2	...	4	7	13	14	
16	Sialkot	68	44	15	6	6	2	5	...	...	1	1	...	...	...	...	...	2	...	1	...	4	21	21	...	
17	Gujranwala	2	22	24	27	...	...	...	...	...	...	...	...	...	...	2	...	2	3	1	...	...	...	3	3	
18	Gujrat	37	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	...	...	...	...	1	10	
19	Shahpur	...	...	...	...	2	...	...	...	...	...	...	...	...	...	...	...	3	...	...	...	1	3	...	...	
20	Jhelum	6	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	2	2	...	
21	Rawalpindi District ...	4	3	26	...	...	3	5	...	...	...	...	...	...	...	...	3	..	...	8	2	...	...	1	...	
22	Rawalpindi Town ...	7	25	11	6	7	3	1	1	3	...	2	6	8	30	38	20	41	52	74	87	101	45	59	22	
23	Attock	1	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	1	12*	1	...	...	* Localized pneumonic outbreak.
24	Mianwali	Never severely infected.											...	...	...	...	...	...	...	...	...	...	...	...	...	
25	Montgomery	...	...	...	3	...	...	...	...	...	...	...	...	...	...	2	4	...	...	2	...	2	8	14	...	
26	Lyallpur	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	3	...	...	...	...	...	11	3	4	
27	Jhang	1	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	
28	Multan	Hardly ever severely infected.											...	...	...	...	...	...	...	...	...	...	1	3	1	
29	Muzaffargarh	Never severely infected.											...	...	...	...	...	...	...	...	...	...	...	...	...	
30	Dera Ghazi Khan ...	Never severely infected.											...	...	...	...	...	...	...	...	...	...	...	...	...	
31	Patiala State	21	2	...	...	...	...	...	...	...	...	...	...	...	...	...	...	3	3	...	2	29	8	4	28	
32	Kapurthala State ...	..	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	5	...	9	6	
33	Jhind State	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	5	2	2	1	2	...	...	

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STATEMENT II.

(*a*) *Plague persisting through quiescent period and extensions therefrom.*

Places where signs of plague were apparent throughout.

1. Rawalpindi ... Rawalpindi district. Population 47,000. Continuous epidemic from May onwards (see statement I).

2. Mallupota ... Jullundur district. Mild epidemic April, May, June ; rat mortality only July, August, September, October, plague case 17th November, 16 others up to 15th December.

3. Narot ... ... Gurdaspur district. Epidemic April and May ; rat mortality noticed in June, July, August and October, plague case 5th November, followed by 5 others.

(*b*) *Imported cases from* (*a*) *not leading to epidemic.*

The following places received imported cases, usually single, but occasionally two or more, total 46, no epidemic followed.

Rawalpindi district	...	Gangal, Mohra Danual, Gujar Khan, Muradi, Mohra, Lalial, Donetala, Dora Budhal, Chak Hira Singh, Mangot, Dhok Ghangla, Malakpur, Machhia and Murree.
Jhelum district	...	Padshahan, Pind Dadan Khan and Jhelum town.
Attock district	...	Kolian Gohru, Jand and Hassan Abdal.
Gujrat district	...	Lala Musa.
Shahpur district	...	Shahpur, Miani and Bhera.
Gujranwala district	...	Gujranwala town, Wazirabad, Eminabad and Jaura.
Sialkot district	...	Sialkot town, Kalawala and Begowala.
Lahore district	...	Lahore City.
Gurgaon district	...	Behora Kalan and Jatauli.
Delhi district	...	Delhi City.

All the above were imported from Rawalpindi City.

(*c*) *Importation from* (*a*) *leading to epidemic.*

Place.	District.	Date.	Source.	Cases and deaths to 15th December.	
Saman	Attock ...	1st November 1911	Rawalpindi ...	11	11
Khushab	Shahpur ...	25th November 1911	Ditto ...	4	4
Kadirabad	Gujrat ...	8th December 1911	Ditto ...	10	10
Kaleran	Jullundur ...	13th December 1911	Mallupota ...	2	1

STATEMENT III.

(*a*) Sporadic cases of plague occurring during the summer and autumn with no subsequent signs of infection.

Serial No.	Plac	Population.	District.	Date.	Remarks.
1	Dali	350	Jullundur	1st August	Four cases April, May, June, rat mortality noticed July, evidently the last case of this epidemic.
2	Kailar	1,050	Ambala	Ditto	Epidemic in spring, last case 19th March 1911.
3	Maloya	1,194	Ambala	5th August	Epidemic in spring, last case March 1911.
4	Dinpur Kalan	...	Gurdaspur	6th August	Ditto
5	Magar	141	Ambala	15th August	Ditto last case April 1911.
6	Daulonangal	766	Amritsar	22nd August	Epidemic 24th April to 13th June, four deaths in this family.
7	Niwan Chand	123	Sialkot	11th September	Rats died in May.
8	Ramgarh Sibian	...	Ludhiana	12th September	.....
9	Sonkhnia	392	Sialkot	20th September	Spring epidemic.
10	Morinda	4,230	Ambala	? September	Epidemic in spring ; last case April 1911.
11	Patiala City	47,000	Patiala State	11th October . , ...	373 cases winter and spring.
12	Butala	2,793	Amritsar	Ditto	Epidemic 8th March to 7th June.
13	Bagraul	698	Patiala State	22nd October	.....

(All single cases except Maloya where there were two.)

(*b*) Sporadic case apparently connected with above.

A man belonging to Chagra class (they apply leeches) attacked with plague on 3rd November at Sattiala, about 3 miles from Butala and it is possible he attended the case in the latter place.

STATEMENT IV.

INFECTION IMPORTED FROM OUTSIDE THE PROVINCE.

(*a*) Imported cases not followed by epidemic.

Serial No.	Place.	District.	Date.	Sources of infection.
1	Hodal	Gurgaon	7th September	Bhopal.
2	Hodal	Do.	23rd September	Do.
3	Jando Singha	Jullundur	29th October	Dehra Dun.
4	Nawazpura	Patiala State	6th November	Hyderabad.
5	Narnaul	Do.	11th November	Do.
6	Lala Musa	Gujrat	2nd December	Bhilsa.
	(*b*) Importation followed by epidemic.			
1	Balipur	Sialkot	20th October	Jammu State.
2	Chak Pindra	Do.	26th October	Do.
3	Khoji Chak	Do.	3rd November	Do.
	(*c*) Spread of infection from (*b*).			
1	Kundal Khalka	Sialkot	9th November	Chak Pindra (epidemic).
2	Kachi Nand	Do.	11th November	Balipur (do.).
3	Dhallewali	Do.	23rd November	Do. (do.)
4	Phuklian	Do.	3rd December	Do. (imported case only).
5	Darya Biddar	Do.	10th December	Dhallewali.

A small patch of localized infection very typical of spread from village to village, see Map B.

STATEMENT V.

REAPPEARANCE, UNCONNECTED WITH IMPORTATION, AND SPREAD THEREFROM.

(*a*) Places where plague reappeared in epidemic form with no evidence of importation at the time.

Serial No.	Place.	District.	Rat mortality.	First case.	Cases up to December 15th.	REMARKS.
1	Basao Kot	Gurdaspur	.....	22nd September 1911	9	4 cases in June.
2	Shahpur Jagan	Do.	29th September 1911	10th October 1911	30	30 cases 10th May to 6th June.
3	Bunga Sahib	Montgomery	Some time before ...	12th October 1911	11	Plague in many neighbouring villages in spring.
4	Khajirka	Gurgaon	1st October 1911 ...	14th October 1911...	55	No cases in spring, bad epidemic in neighbouring village.
5	Samana	Patiala State	.....	Ditto ...	3	123 cases 11th February to 27th July.
6	Dugal	Ditto	.....	Ditto ...	3	Neighbouring villages infected in spring.
7	Chak 370	Gujranwala	3rd October 1911 ...	18th October 1911...	6	No history of infection in spring.
8	Masrur	Gurdaspur	15th October 1911...	22nd October 1911	11	18 deaths, 24th April to 3rd June.
9	Ghaga	Patiala State	.....	Ditto ...	4	.....
10	Umra	Hissar	1st October 1911 ...	24th October 1911	68	Rat mortality in May.
11	Ghagon	Hoshiarpur	6th October 1911 ...	25th October 1911	10	2 cases imported in March, neighbouring villages infected in spring.
12	Mullana	Ambala	11th October 1911...	26th October 1911...	25	Epidemic in spring.
13	Narah...	Karnal	22nd October 1911	Ditto ...	11	Incomplete epidemic, 18 cases between May 2nd and 29th May 1911.
14	Pakasma	Rohtak	Some time before ...	Ditto ...	89	1st May, 12th June 1910, recrudescence 9th October 1910 to 13th January 1911.
15	Chaiali	Patiala State	.....	28th October 1911	19	Neighbouring villages infected in spring.
16	Saincha	Gurdaspur	18th October 1911	1st November 1911	5	Ditto ditto.
17	Ghabdan	Jhind State	.....	Ditto ...	12	.....

Serial No.	Place.	District.	Rat mortality.	First case.	Cases up to December 15th.	Remarks.
18	Ghandwan	Ludhiana	7th November 1911	11th November 1911	27	Spring epidemic, last case 5th May.
19	Balu	Patiala State	18th October 1911	3rd November 1911	29	29 cases, 29th February to 18th June.
20	Bham	Hoshiarpur	.....	4th November 1911	3	Epidemic ended 14th July, infection reappeared same part of village.
21	Kalas	Lahore	.....	6th November 1911	6	Two cases in April, suspicious case 22nd October. Small out of the way village.
22	Lodhianwala	Gujranwala	3rd October 1911 ...	8th November 1911	17	26 deaths, 18th April to 31st May.
23	Naggal	Ambala	One month previous	Ditto ...	30	Spring epidemic, last case 24th March 1911.
24	Mansurwal	Kapurthala State	.....	10th November 1911	5	Infected 23rd February to 22nd March.
25	Gujarwal	Ludhiana	7th November 1911	11th November 1911	49	Spring, 8 cases, 3rd May to 5th June 1911.
26	Sadhura	Ambala	3rd November 1911	18th November 1911	34	Spring incomplete epidemic, last case 25th April 1911.
27	Chak 109	Lyallpur	Some time before ...	20th November 1911	...	Spring infection, 10 cases 27th May to 3rd June.
28	Bhukan	Montgomery	Ditto ...	20th November 1911	24	Spring infection, suspicious case 14th August near villages infected in spring.
29	Bua	Gurdaspur	1st November 1911	21st November 1911	3	Infected April and May.
30	Raowal	Ferozepore	In September and October.	22nd November 1911	2	Spring epidemic, suspicious case end of October.
31	Miani Bakarpur	Kapurthala State	.....	1st December 1911	9	Incomplete epidemic 5th to 19th April.
32	Dhiroke	Sialkot	25th November 1911	2nd December 1911	4	No spring infection here, nor in near villages.
33	Bakarwala	Delhi	.....	2nd December 1911	6	Imported cases in April, no previous epidemic.
34	Dulasinghwala	Ferozepore	.....	5th December 1911	4	No infection in spring.
35	Akhbarpur	Kapurthala State	Few days before ...	10th December 1911	12	No infection previous. Spring State infected.
36	Kotli Chahm	Sialkot	10th December 1911	10th December 1911	1	Sporadic case? No history.
37	Mahal Gahla	Jullundur	.....	14th December 1911	3	245 cases, 23rd February—7th May.
38	Shujabad	Multan (not shown on map)	.....	30th November 1911	5	Four cases in May in one family, the case of 30 hours occurred in an adjacent house.

(*b*) Imported cases from (*a*) not leading to epidemic.

This group contains only one town : Alawalpur in the Jullundur tahsil. One case was imported from Khajirka ¾ mile distant.

(*c*) Epidemic following importation from (*a*).

Serial No.	Place.	District.	Rat mortality.	Cases up to December 15th.	Remarks.
1	Ram Bateri	Gurdaspur	2nd October 1911 ...	3	From Basaokot (*a*) 1.
2	Chak 585	Lyallpur	19th October 1911 ...	3	Possibly from Bunga Sahib (*a*) 3, no definite history.
3	Rohti	Karnal	27th October 1911 ...	11	From a village in Patiala State, no definite history.
4	Mohri	Karnal	8th November 1911 ...	6	Possibly from Rohti (*c*)3.
5	Kamalpur Jattan	Gurdaspur	12th November 1911 ...	5	From Basao Kot (*a*) 1.
6	Barwala	Ambala	20th November 1911 ...	1 and rat mortality.	From Nangal (*a*) 23.
7	Thaska Miranji	Karnal	20th November 1911 ...	4	From Rohti (*c*) 3.
8	Bhalot	Rohtak	20th November 1911 ...	5	From Pakasma (*a*) 14.
9	Bachhli	Gurdaspur	26th November 1911 ...	3	From Masrur (*a*) 8.
10	Lahouri Khurd	Ludhiana	29th November 1911 ...	10	From Ghundwan (*a*) 18.
11	Anandpur	Hoshiarpur	4th December 1911 ...	8	Possibly from an Ambala village, but the evidence is in favour of no importation.

PART II.

THE IMPORTANCE OF ACTUAL INVESTIGATION.

No one who has watched the beginnings of the annual epidemic in the Punjab year after year can fail to observe that the reappearances of epidemic plague in a short space of time, widespread over a large area in places widely separate and remote from each other, is entirely different to what occurred in the original diffusion of infection throughout the province from district to district by importation. A study of the figures alone without the check of analysis and investigation of each separate infected locality must inevitably lead to faulty inference and erroneous conclusion, for although it is true that the provincial figures nearly always show a continuance of plague cases throughout the quiescent period, they will not show that this is not a connected sequence but merely the overlapping of widely separate and unconnected infections, a fading epidemic in one part of the province and a reappearance in another perhaps hundreds of miles distant. The same is true if the provincial figures are separated into district figures and each separate district studied; no inference or conclusion of any value can be deduced unless the figures are studied in the light of actual investigation of the epidemiological facts. Such investigation has now been going on for many years and each succeeding year's experience supports and confirms past observation, going back to the first introduction of plague into the province, when some months interval separated the importation of infection from the first appearance of epidemic plague which resulted from it, and to those early years when plague was confined to a small area under very close observation and plague was found to reappear annually although the area had apparently been free for months (see Appendix, example 7).

With regard to the actual value of the evidence it may be said that epidemics have been investigated by a medical staff experienced in plague work, with a knowledge of the country and the people and also of the difficulties inherent in such examinations. It is sometimes objected that plague is frequently concealed and that such investigations as these are consequently valueless. The objection is not a valid one, for although plague may not be reported for some time, with a touring staff it will nearly always come to light and an examination on the spot will elicit the true facts; plague is reported, the village is visited, the dates of plague attacks and of the appearance of rat mortality are ascertained, both of the present and the preceding spring infection, there is no history of any plague occurrence in the interval, it is not reasonable to suppose that signs of plague in the summer have been deliberately concealed while those of the spring and autumn are admitted. It must also be remembered that in villages where most of these investigations have been carried out, especially in the smaller ones, the movements of individual inhabitants and the arrival of guests and strangers are very accurately known and consequently connection or otherwise with infected localities is usually fairly easy to establish.

It is upon the accumulated observation of many years that the following remarks and conclusions are based.

The origins of the annual epidemic.

Infection persists throughout the quiescent period in varying numbers of places every year, and such persistence is sometimes marked by a continuous epidemic, such as Rawalpindi City, 1911, or by rat mortality only, either continuous or at intervals, as in Mallupota and Narot villages, 1911; more frequently there are no such signs and epidemic plague reappears after an interval of complete apparent freedom, with no fresh importation to account for it. The annual visitation originates from both these classes, persistent infection marked by actual visible signs, and persistent infection which is not so marked, and, of the two, the latter are far more common than the former, and, in addition, are much more dangerous, for whatever may be the local conditions that determine the persistence of human plague and visible rat mortality throughout the summer, such visible persistence is evidence of a much more serious disturbance of the rat population than that

which is occurring in places where persistence of infection is not so marked, and the more severely the rat population is affected during the summer, the less likely is such an infection likely to extend to a serious epidemic when conditions in the autumn become favourable for plague activity. These centres, where no infection has been apparent during the summer, therefore form by far the most important origins of the annual epidemic, not only because they are much more numerous but also because they are much more likely to suffer severe epidemics and consequently be more dangerous diffusion centres than places where epidemic and epizootic plague are evident through the quiescent period.

The infection precedent to these reappearances unconnected with fresh importation.—In the absence of fresh importation, the reappearance of plague in a locality can only be attributed to a previous infection although no signs of plague have been apparent in the interval between; such previous infections may vary greatly in extent and this variation affects both the extent of and the time and manner of appearance of the subsequent reappearance. The previous infection may be marked by :—

(*a*) a complete epidemic, that is to say one where infection has run completely through and temporarily exhausted the rat population of a locality and which has come to an end with this exhaustion ;

(*b*) an incomplete epidemic, where the epidemic has been brought to a conclusion by hot weather conditions before infection has passed completely through the rat population ; this may vary from an epidemic of considerable proportions to only one or two cases. [See statement V (*a*), and Appendix, cases 19 to 24] ;

(*c*) rat mortality only. (See Umra, Part 1, page 7, and Appendix, cases 25 and 27) ;

(*d*) cases occur where it is evident that infection must have passed to the rat from an imported case, although no rat mortality was observed at the time, Bakarwala, page 7, is an example of this. (See also Appendix, cases 26 and 28) ;

(*e*) in the last two classes the introduction of infection is marked by an imported case, but it is recognized that infection may be imported without the carrier suffering, consequently there may be absolutely no signs whatever of the introduction of infection in the spring. Instances where this appears to be the only explanation are fairly common, the first appearance of epidemic plague in a district in the autumn being in a village apparently unaffected in the spring, but near an infected village, Khajirka, page 6, Part 1, is a specific instance of this. (See also Appendix, cases 29, 30 and 31.)

In those cases where the epidemic is incomplete, reappearance is likely to appear early the following season. The smaller the infection in the spring, the less the rat population will have been affected and the more likely that the appearance will develop into a severe epidemic.

When the spring infection is small and localized, plague often reappears in the same locality, see Bakarwala, page 7. (See also Appendix, cases 25, 26, 27, 28, 32, 33, 34, 35 and 36.)

When the previous infection is complete, reappearance if it occurs will not usually appear in the autumn but later on when the rat population has had time to recover.

Reappearance, unconnected with fresh importation is not confined to the autumn.—The special investigations that have been carried out in 1907 and in 1911, have been confined to the autumn because it is then, when the plague season is a mild one and only beginning and when infections are few and far between, that thorough examination can be made and the fact of intercourse between infected localities wide apart can be more definitely denied. In all our observations wherever intercourse between villages has seemed probable or even possible the infection has been credited to importation although there may

have been no definite evidence of the fact With a gradually increasing number of infections the fact of some intercourse, however slight and possible, importation becomes more and more difficult to exclude.

There is no doubt, however, that plague continues to reappear, with no importation to account for it, throughout the plague season and it is probably not less common in the spring than in the autumn, and although in the former it is difficult to exclude the importation factor, the manner in which plague reappears often seems to be sufficient evidence that the reappearance is not the result of fresh importation. When a village has suffered a complete epidemic the previous season and the rats have suffered throughout the village, the inference is natural and sound that if plague reappears apart from fresh importation, such reappearance will not be confined to a single spot from which the epidemic spreads but will occur in more than one spot and in an apparently indiscriminate manner entirely different to the definite spread of infection after an imported case. No one who has much experience in the field can fail to note this very marked difference between the two classes of epidemics, those due to fresh importation and those which are not ; the village of Dhand which was under the close observation of the Plague Research Commission is an example of what is meant but much more definite examples are frequently met with and it is not uncommon to find that the reappearance of plague consists in the *simultaneous* occurrence of two, three or even more plague cases in separate parts of a village between which no connection can be traced (See Appendix cases 37, 38, 39, and 40). Even when epidemic plague is present in neighbouring villages this is extremely strong presumptive evidence that the cause of the reappearance is not importation, and when it occurs in a village far from other infections and with no evidence of intercourse, the assumption that importation has been effected from some unknown source into two or more parts of the same village appears not only untenable but ridiculous.

Conditions favouring reappearance unconnected with importation.— Those conditions which are favourable for plague activity generally are also favourable for these reappearances ; for instance the summer and autumn of 1911, with a late and scanty monsoon was unfavourable for plague, epidemics were as a rule much milder than usual, there was much less spread by importation, imported cases were less frequently followed by indigenous cases and reappearance without importation was less common. There is no doubt that the factor of humidity which is so necessary for plague activity also encourages such reappearance ; in the autumn of 1911 plague without importation to account for it appeared principally in the southern part of the Punjab where good though late rains fell in September, while the central Punjab, Lahore, Amritsar etc., usually a hot bed of plague, escaped. Such reappearance is more common in highly irrigated areas, in places which have a high subsoil water level, and in the submontane tract. Speaking generally it may be said that tracts that suffer from bad epidemic plague are most liable to such reappearance.

The local factors determining the phenomenon are probably extremely complex ; one is certainly the condition of the rat population, for plague rarely appears in the autumn in a locality which has suffered a complete epidemic in the beginning of the year. Flea prevalence no doubt is a determining factor but such prevalence is very variable and although there is a general seasonal variation, there may be great differences in different years and in different parts of the province and moreover there may be marked differences in flea prevalence even in two villages in the same year as was the case in Dhand and Kasel in 1907 ; one may go a step further and presume that such differences may occur in different parts of the same village ; the part played by this factor is impossible to estimate, but it is probable that the almost haphazard reappearance of plague unconnected with fresh importation is to be explained by its extreme variability. There is no doubt that insanitary villages and areas of towns are particularly liable to such reappearance, and some particularly dirty villages where plague reappears nearly every year have been called endemic centres ; certain parts of some towns have an unenviable reputation in that plague reappears frequently in these places.

Reappearance of plague without importation does not inevitably follow a previous epidemic.—It has never been asserted that epidemic plague will occur in every place infected during the previous season, indeed this is very far from being the case; probably only quite a small proportion of villages suffering from an incomplete epidemic in the spring show a reappearance of plague in the autumn and this is of course particularly true in a mild plague season such as the autumn of 1911. From the work of the Plague Research Commission the most reasonable explanation of the persistence of infection throughout the quiescent period is that an epizootic of acute plague continues below ground, but owing to a scarcity of fleas, at a very slow rate of progress, the rat mortality being so little as not to appear above ground and, with an increased flea prevalence determined by both general and local conditions, the epizootic quickens up, rat mortality becomes visible and the epidemic begins; this assumption would adequately explain the apparent haphazard reappearance of plague for, with but few fleas and a very slow epizootic, it is reasonable to suppose that the chain of infection must be very liable to be broken with complete cessation of the rat disease and consequently no subsequent outbreak; the chain would be less likely to be broken in places where damp, dark and dirty conditions favour verminous infestation, places where plague is particularly liable to reappear; the explanation of the non-recurrence of plague in large towns is probably that the chain of infection is more likely to be broken where buildings are *pukka* and rat colonies more definitely separated than in mud-built villages. The only difficulty in my own mind in accepting absolutely this explanation of the persistence of infection by a slow but continuous epizootic is found in those cases such as Bakarwala quoted above, where indigenous plague appeared in the same house seven months after infection was imported; is it possible for an acute epizootic however slow to continue over such a long period in a single rat habitat?

However that may be, the fact remains that in a large proportion of places infected in one season, infection does not persist and epidemic plague will not reappear unless fresh importation is effected.

Importation.—It has never been contended that plague reappears in every locality infected the previous season, nor would any one attempt to deny that importation is any less potent now as a factor in the spread of infection and the production of the annual epidemic than it was when the disease originally spread over the province by this method. The annual visitation is the compact of the two factors, reappearance not due to importation, and importation, and of these two, it is highly probable that the majority of local epidemics is to be ascribed to the latter; the importation of infection, however, presupposes some infected place from which the infection is derived, *i.e.*, the origins of the epidemic, and in the Punjab the large majority of these original epidemics from which plague spreads in the usual way by importation is caused by the reappearance of epidemic plague with no importation to account for it. A very small part of the annual epidemic is caused by the spread of infection from those places where plague infection has visibly persisted through the summer, the greater part is caused by reappearance unconnected with importation and the spread therefrom by importation. If it were possible by any means to absolutely prevent the spread of plague by importation, the annual epidemic would be enormously reduced but if such reappearances could be prevented a still greater reduction would be effected, and plague would soon be a thing of the past. A map is attached, map C, which shows the reappearance of infection and spread by importation therefrom in the district of Ludhiana, the worst infected district in 1911-12; every possible allowance has been made for inter-village intercourse and in many cases there is no definite evidence of importation but only a conjectured possibility, all such however have been marked as importation; it will be seen that the large majority of epidemics were due to imported infection, but it is clear that none of these would have occurred had plague not reappeared in certain places, (the importations from outside were also from epidemics, the originals of which were unaccounted for by importation).

The practical importance of the subject.—The reason why so much attention has been directed to this phenomenon is that its recognition is of vital importance if any attempt is to be made to control the annual epidemic. The plague season

which has its beginnings in the autumn continues for a determinate period of time up to a certain date, usually in May, when the maximum is reached and decline begins; every week therefore that the beginning of the epidemic can be delayed means a corresponding shortening of the epidemic and *the saving of a week's mortality at the height of the plague season.* In the Punjab therefore for many years the primary objective of the plague department has been the attack on the origins of the epidemic because, for every place where the reappearance of plague is prevented or a commencing infection stopped, a large saving of life may be computed in-as-much as not only the primary epidemic but a number of secondary epidemics caused by importation from the first will be avoided. This attack has been the foundation of the Punjab plague policy and looked upon as of even greater importance than the saving of a certain number of lives by inoculation, evacuation, etc., when the epidemic is raging and control has been lost.

It is the fact that plague may reappear without any fresh importation after a considerable interval of apparent freedom that has made this problem one of supreme difficulty. It will be conceded that if, as at one time it was thought, the only dangerous places from which the annual epidemic arose were those where actual signs of plague infection persisted throughout the hot weather, the problem would be comparatively simple, for these places are as a rule so few in number that it would be legitimate to apply such compulsory measures as would effectually prevent any spread of infection therefrom. But it has long been evident that, however perfectly such measures were carried out, only a small proportion of the annual epidemic would be prevented; it therefore became necessary to consider not only those few places marked by rat mortality and human plague but a very much larger number, potentially dangerous in which reappearance was probable, though not in any way inevitable. That the number is very large has been demonstrated in this paper, not only all places suffering an incomplete epidemic in the spring and all particularly dirty and insanitary places which annually suffer, but also places themselves apparently uninfected in the spring where it was possible that infection has been implanted in the spring from neighbouring villages. For the attempt to prevent these epidemics, the origins and the annual visitation, there was only one measure that could be applied because it was the only one that the people would generally accept in the absence of plague and that measure was rat destruction. That a reduction in the rat population could be effected by trapping and poisoning had been established, and with our present knowledge it must be conceded that a reduction in the number of rats must lessen the possibility of a reappearance of plague, or if plague should reappear, must mitigate the severity of the epidemic; rat destruction was therefore widely used as the only means at our disposal by which we could hope to affect the origins of the annual epidemic; rat poisoning alone was also used as a means of rapidly, though temporarily, reducing the danger of epidemics in healthy villages in particular danger of importation owing to their proximity to infected ones. That rat destruction has taken such an important position in the Punjab is due, not to its general haphazard application in the vague hope of doing some good, but in the pursuance of this very definite objective, the suppression or mitigation of the origins of the annual visitation. It is unfortunate, though inevitable, that the effect of this campaign that has been waged for so many years can never be accurately gauged or the life saving expressed in figures, but it is probable that it has been considerable and though this is no place for the consideration of this problem, it may be permissible to refer to one little fact recorded in the first part of this paper, that rat destruction was carried out in the Delhi area in the autumn of 1911 in fourteen places where rat mortality had begun and in none of these had epidemic plague appeared up to the end of the year.

A final glance at map C. will show that the annual epidemic may be controlled in two separate ways, first, the suppression of the origins, second the prevention of spread by importation therefrom into healthy villages; it is to the first of these that effort has been particularly directed in the past for it has been impossible to stop the entry of persons into healthy villages, and equally impossible to generally apply any known method of disinfection of clothing and property; recently, however, the power of the sun's rays for this purpose has been demonstrated and with such a disinfectant universally available, costing nothing and easy of application, there is hope that in the future the second line of action will have its effect in controlling the epidemic and reducing the annual death rate.

APPENDIX.

Summary of the results of the investigation of the origins of the 1907-1908 epidemic with details of certain infections.

(From "The Recrudescence of plague," Bombay Medical Congress Transactions January 1909.)

The plague season 1907-1908 was a mild one doubtless due to the failure of the autumnal monsoon 1907; infections were few in the first part of the season and circumstances favourable for investigation,

Every infection in twelve selected districts was carefully investigated up to the time when rapidly increasing numbers of epidemics rendered it impossible.

In these twelve districts, out of a total of 277 local epidemics investigated, eleven or 4 per cent. were instances of persistence of infection, apparent more or less throughout the quiescent period; from these eleven, infection was traced to only four others, a total of fifteen.

In 126 cases, or 45·5 per cent. plague reappeared without any traceable importation to account for it. As a result of this investigation and previous experience the following conclusions were formulated:—

(*a*) Recrudescence tends to appear early the next season in those places which have suffered only incomplete epidemics in the previous one, that is to say, where the epizootic has been cut short by the hot weather before affecting the whole of the rat population.

(*b*) An incomplete epidemic is not a necessary precursor, for importation may be effected late in one plague season without any epidemic or even any signs of epizootic until, after an interval of apparent freedom, recrudescence occurs.

(*c*) Recrudescence following complete epidemics, where the rat population has been completely dealt with before the hot weather, more generally appears late in the following plague season, presumably because time for the recovery of the rat population is necessary.

(*d*) The severity of the epidemic accompanying the recrudescence will vary directly with the incompleteness of the previous one; the less the rat population has been affected during the first epidemic the more severe will be the following one.

(*e*) Generally, therefore, recrudescence occurs earlier and is more severe after incomplete than after complete epidemics.

(*f*) The more insanitary a locality is the more liable it will be to recrudescence. The reappearance of infection usually occurs in the most insanitary part of a locality, in houses, dark, damp and ill-ventilated. Briefly, all conditions favouring rat and flea infestation are favourable for recrudescence.

EXAMPLES.

1. Illustrating the reappearance of plague following incomplete epidemics and not connected with importation.

CASE 19. Village Makhanwindi, Amritsar tahsil, Amritsar district. Twenty cases occurred between 27th March and 28th June, 1907; slight rat mortality was noticed after the last case. The first case of the next epidemic occurred on 23rd October, 1907, preceded for two weeks by rat mortality; no evidence of importation; no connection with any infected village. Interval 3⅓ months.

CASE 20. Village Dawlonagal, Amritsar tahsil, Amritsar district. Five cases between 11th May and 1st June, 1907. First case of next epidemic October 21st, 1907, preceded by rat mortality for ten days; no history of importation; the only infected village in the district at this time. Interval 4⅓ months.

CASE 21. Village Thandi Serai, Jullundur tahsil, Jullundur district. Four cases between 9th May and 24th May, 1907; the next case occurred on 4th January, 1908; no evidence of importation; the only infected village in the district at this time. No rat mortality was observed. Interval 7½ months.

CASE 22. Village Kilchpur, Kharian tahsil, Gujrat district. Thirty-eight cases between 17th May and 12th June, 1907. Next case on 16th April, 1908, preceded by rat mortality for ten days, no evidence of importation; only one other village infected in the district, which was also due to recrudescence. Interval 10 months.

CASE 23. Village Sojan, Dasuya tahsil, Hoshiarpur district. Eight cases between 9th May and 18th June, 1907; the next case occurred on 16th November, no rat mortality was observed but a bad smell was noticed in the houses where recrudescence appeared. No evidence of importation and there were no infected villages near. Interval 5 months. Interesting in that it shows that although rat mortality was not apparent it probably was occurring.

CASE 24. Village Nakki Brahmanan, Shakargarh tahsil, Gurdaspur district. Four cases following importation occurred between 4th May and 1st June, 1907; the next case on 17th January, 1908, preceded by rat mortality for a fortnight. No evidence of importation; no connection with infected villages. Interval 7 months.

2. Reappearance following importation the previous spring without any epidemic.

CASE 25. Village Ratiya, Fattehabad tahsil, Hissar district, population 3,383. A case of plague was imported into this village from an infected village in Patiala territory on 23rd April, 1907, rat mortality began a few days after the arrival of infection; only one other case occurred, on 1st June, rat mortality continuing for a few days after. The next case occurred on 9th August preceded by rat mortality for 15 days. The interval, when there was no apparent rat mortality or cases, was quite a short one, about one and a half months. There was no evidence of importation to account for the rat mortality or the case of August 9th, but the latter occurred in a house quite close, though not attached by common walls to the house where the case occurred on 1st June. This epidemic lasted till December 4th, with a total of 66 cases and 44 deaths. After another free interval of nearly 3 months another case, preceded by rat mortality for 10 days, appeared on 10th March, a small epidemic of 5 cases resulting between this date and 21st March. No evidence of importation to account for this second epidemic. It is interesting to note this double recrudescence in one plague season, each occurring at the time when plague is most active, the autumn and the spring.

CASE 26. Village Daryapur, Fattehabad tahsil, Hissar district. A small village of 357 inhabitants. A person, suffering from plague, came from an infected village in Patiala State on the 1st April, 1907, and died. No rat mortality was noticed in connection with this case at the time, but about the 6th September slight rat mortality was noticed in a house adjoining the one in which the original case stayed, and on 22rd September a plague case occurred in this house; there was no history of importation to account for the rat mortality or the case. The epidemic resulted in 16 cases, ending on 15th October. The interval between the importation and appearance of infection was more than five months.

CASE 27. Village Tohana, Fattehabad tahsil, Hissar district, population 5,931. Between 22nd February and 20th May, 1907, 17 cases were imported into this village which appears to have been looked upon as a safe haven by adjacent infected villages in Patiala State. All these cases were removed outside the village in less than 24 hours after their arrival except one, Chumnia, a *mahajan*; in his house a dead rat was observed in May and he immediately evacuated; slight rat mortality was noticed in connection with this but no indigenous cases occurred. About the 3rd October rat mortality began in a house, close to that of Chumnia, and in 3 or 4 others which had common walls with the first; the first case occurred in this house on 13th October. There was no evidence of importation to account for the infection. The epidemic was a very

small one ending in the second week of November with a total of only five cases. The interval was at least four months. This village was again infected in the spring by importation, some guests came from an infected village, Chagli, in Patiala State, on 20th February, 1908; rats began to die a few days later in the house in which they stayed and on the 5th March, a case of bubonic plague appeared; this epidemic was also a small one of only ten cases in all, the last occurring on 7th April.

CASE 28. Village Juggian Kokaran, Sharakpur tahsil, Lahore district. This is a very small village of only 40 inhabitants, living in 5 new houses. A case of plague was imported on 15th April 1907, dying a few days later, no rat mortality was noticed in connection with it. At about the end of December dead rats were found in this particular house and the first case occurred on January 23rd, 1908, followed by twelve others; no evidence of importation, and this village was the only one infected for many miles round. Interval about 8 months.

3. Autumnal epidemics following unmarked importation in spring.

CASE 29. Village Rayanwala, Naraingarh tahsil, Ambala district. There had never been any plague in this village before, but a neighbouring village Sahla, with which the inhabitants of Rayanwala have free communication, was infected in the spring of 1907, the last case occurring on April 24th. Rat mortality was noticed in Rayanwala in May but no human beings developed the disease. Nothing further was noticed till October 15th when rat mortality again became apparent and the first indigenous case occurred on October 23rd. There was no evidence of importation to account for this autumn infection and the only explanation that seems possible is that although no human case occurred importation was effected in the spring from the neighbouring village. The interval was some four or five months.

CASE 30. Village Motaliwala, Muktsar tahsil, Ferozepore district. There was no plague in this village in 1906-07, but neighbouring villages were infected late in the spring. There were no signs of the disease until January 22nd, 1908, when a plague case occurred preceded by rat mortality for five days. No evidence of any importation and no connection with any infected village in the autumn. Interval indefinite but at least some 6 or 7 months.

CASE 31. Mianwali district, except for a few imported cases not leading to epidemics, was practically never infected till the spring of 1907, when plague was imported into a village, Chokrala, and an epidemic followed causing 67 deaths, the last case occurring on 6th July; this was the only infected village during this plague season. Towards the end of April, 1908, the district being meanwhile absolutely free of plague since 6th July 1907, rat mortality was noticed in Kund, a village 3 miles from Chokrala, followed by the first case of plague on April 30th. There was no history of importation and no other infection anywhere in this part of the province. The only explanation appears to be that importation must have been effected in the early summer from Chokrala. Interval at least nine months.

4. Showing definite connection by locality between spring and autumn infections.

CASE 32. Village Mirchpore, Hansi tahsil, Hissar district, population 3,161. A case of plague was imported on 27th April, 1907, and 3 or 4 days after rat mortality was noticed; another case was imported on May 5th, and the first indigenous case occurred on May 13th, rat mortality spreading from the first house to those of the same *mohalla*. Only four more people were attacked, the last being on June 14th. About the 30th August rat mortality was observed to be occurring in houses attached to those infected in June, and the first case occurred on September 8th. This resulted in an epidemic of 624 cases, ending on 15th February, 1908. There was enormous rat mortality but the case mortality was extraordinarily low, only 162 cases ending fatally. Interval 2½ months.

CASE 33. Village Narnaund, Hansi tahsil, Hissar district. Plague case imported on 5th June, 1907; a few days later dead rats were noticed in the house; a very slight incomplete epidemic followed resulting in 6 cases altogether,

the last on July 26th. About the 7th September rat mortality began in a house close to those previously infected followed by the first case on September 22nd. No history of importation or connection with infected localities. The epidemic ended on 19th April, with a total of 124 cases and 52 deaths, another instance of a low case mortality. Interval only 1½ months.

CASE 34. Village Chak Santal, population 916, Sialkot tahsil, Sialkot district. A plague epidemic raged here from 25th January to 4th May, 1907, resulting in 45 cases. Nothing further was noticed until the 2nd November, 1907, when a dead rat was found in a house where two women died in May, and the last dead rat of the preceding epidemic was found. Twenty cases and thirteen deaths. No history of importation and the first and only infected village in the district. Interval 6 months.

CASE 35. Sirsa town, population 17,058, Hissar district. This town was infected by importation on 2nd February, 1907, ninety-one cases occurring between that date and the 11th June, the epidemic was confined to one *mohalla* of the town. Nothing further was noticed until the 27th October, 1907, when rat mortality was noticed in a house in the same *mohalla* followed by the first case on 1st November. No evidence of importation. Rat destruction was at once carried out which probably accounts for the mildness of this epidemic which only resulted in 12 cases, the last on 15th January, 1908. Interval 4½ months.

CASE 36. Village Mirzapur, Hissar tahsil, Hissar district, population 1,391. Infection imported on 7th May, 1907, 5 days later rat mortality was noticed, the second case occurring in the same house on 15th May; the epidemic was confined to a few Muhammadan houses, all having common walls with the first one infected, and led to nine cases, the last on 6th June, rat mortality continuing for 15 days after this date. Nothing further was noticed until 24th November, when rat mortality again appeared in a *Chamar's* house which adjoins and has common walls with the Muhammadan house infected in June, followed by first case in the same house on December 4th; rat mortality to a slight extent continued in this same block of houses but no further case occurred till late in January, 1908; it then spread throughout the village and caused 77 cases, the last on 13th April. Interval 5 months.

5. Illustrating reappearance in separate parts of same locality.

CASE 37. Village Wiram, Chunian tahsil, Lahore district, population 923. Seventy cases of plague occurred in this village between 22nd January and 2nd May, 1907; nothing further was noticed until six cases occurred in different parts of the village practically simultaneously on 7th December, 1907. No rat mortality was observed but a bad smell, probably indicative of it was noticed in the infected houses previous to the human cases. No further cases occurred. There was absolutely no evidence whatever of any importation, and there was no communication with any infected village. Interval 7 months.

CASE 38. Village Hudiala, Sharakpur tahsil, Lahore district, population 500. A case of plague was imported into this village on 5th February, 1907, no rat mortality was observed in connection with this case and there were no other cases. Rat mortality began about October 10th scattered about the village, with no definite connection with the previously infected house, and the first three human cases, November 1st, 7th and 9th were in separate parts of the village and apparently quite unconnected with each other. No evidence of importation or connection with infected villages. Interval 8 months.

CASE 39. Village Butala Sharm Singh, Gujranwala tahsil, Gujranwala district, population 1,717. An epidemic causing eighty deaths occurred in the spring of 1907, the first case being on 2nd February and the last on 13th June. No further signs of infection until the 16th February, 1908, when two cases occurred on the same day, one in a house on north side, and the other on east, some sixty houses apart with no connection between the two. No evidence of importation or connection with infected villages, no rat mortality noticed. Interval 8 months.

CASE 40. Town Khudian, Chunian tahsil, Lahore district, population 3,401. An epidemic causing 85 deaths took place between 17th January and 14th

May, 1907. Two cases of plague were imported in December, 1907, but no rat mortality or human cases followed in connection with them. At about the end of February rat mortality was noticed as occurring generally throughout the town and on March 9th, human cases occurred simultaneously in three different streets and the epidemic began. Interval 9 months. The widespread nature of the re-appearance of the disease in this case is indicative of recrudescence, in spite of the fact that importation had occurred previously; it must be admitted however that these importations may possibly have led to the subsequent epidemic.

6. Illustrating reappearance beginning as pneumonic plague.

CASE 43.—Anandpore, population 573. A suburb of the town of Pathankot, Gurdaspur district. 176 cases occurred here between the 11th November, 1906 and 10th April 1907, the epidemic being originated by an imported case which was followed by rat mortality in the usual way. Nothing further was observed until the 25th August when a case of pneumonic plague occurred; the second case was also pneumonic and the next two bubonic, all occurring between the 25th and 30th August. No rat mortality was observed. No history of importation; no other infection in the district, this being the first place to report infection. Interval four and a half months.

CASE 44.—Village Padri, Tarn Taran tahsil, Amritsar district, population 2,463. The last case of a very slight epidemic occurred on 27th April, 1907. Nothing further occurred till 1st February, 1908, when a case of pneumonic plague occurred in the person of a carpenter, living in a house infected two years before, the disease quickly killing him and all his family except his wife and then spreading to the rest of the village. No evidence of importation. No infected villages near. Rat mortality not observed but the reporting officer notes that it probably occurred. Interval 9 months.

CASE 45.—Village Mattoo Bhaiki, Gujranwala tahsil, of the same district, population 1,179. The last case of an epidemic causing 57 deaths occurred on the 7th June 1907. No signs of infection till 16th January, 1908, when a single dead rat was found in a house adjoining the one in which the subsequent case occurred, one of pneumonic plague on 20th January. No dead rats were seen in this house but it was noticed that the rats, which were very numerous, suddenly disappeared two or three days before the case. The second case was bubonic. No evidence of importation, no infected villages near. Interval 7 months.

CASE 46.—Village Papnakha, Gujranwala tahsil, of the same district, population 2,032. An epidemic resulting in 278 deaths ended on 10th May 1907. There was no apparent infection until nearly ten months later when two dead rats were found on the 4th March, 1908, in the house of one Maula Dad who fell ill of pneumonic plague on 14th March. The epidemic was slight, only twelve cases. No history of importation or connection with infected villages. Interval 10 months.

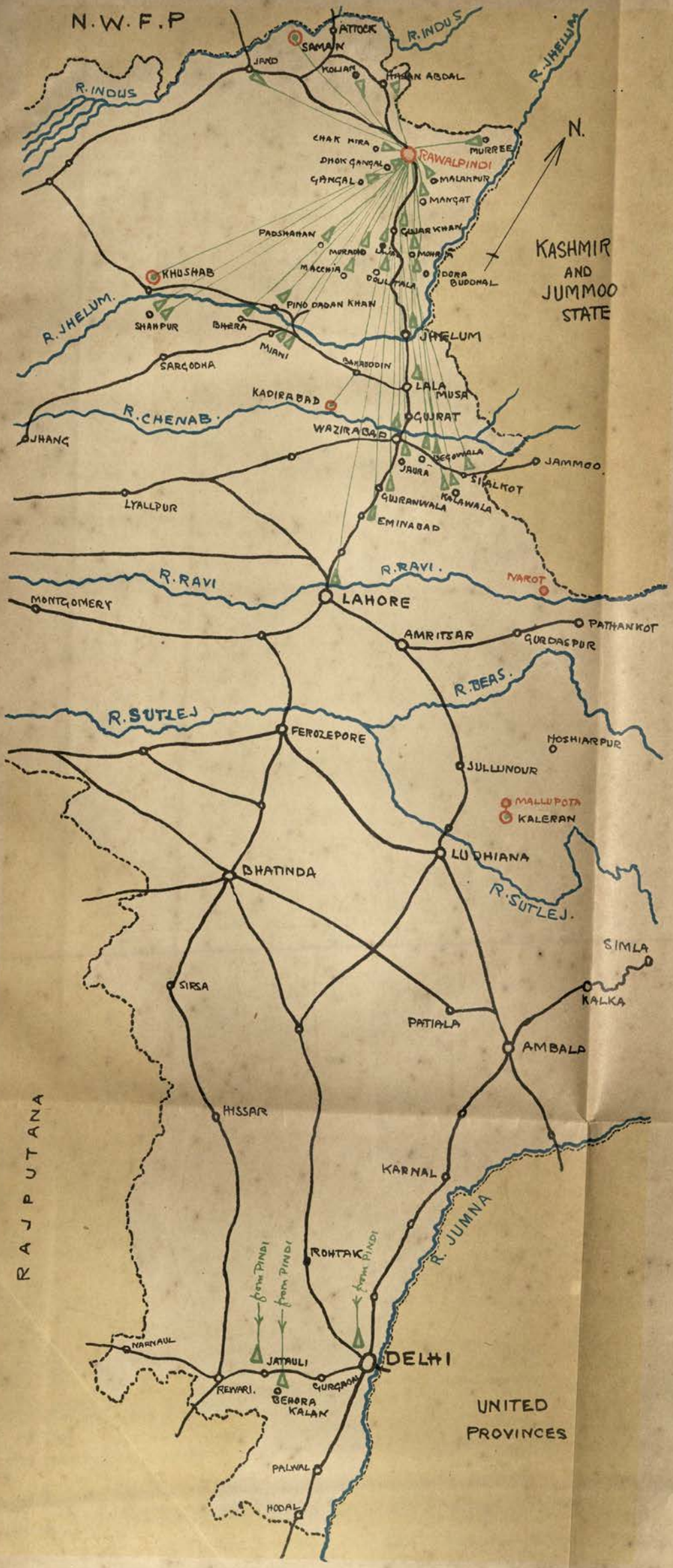
7. Showing corroborative evidence from earlier epidemics.

Even at this distance of time a brief study of the first epidemics in the Punjab, when the area infected was small and under close observation, will be useful. The history of the first importation of plague into the province is an interesting one, and this case belongs to the class where importation did not give rise to other signs of infection at the time, these appearing later after a considerable free interval.

CASE 41. In the year 1897, Ram Saran, returning to his home from Hardwar was taken ill at Rahon town in the Jullundur district, on the way and eventually arrived, seriously ill, at his village Khatkar Kalan in the same district on April 28th, dying on the next day in his small mud-house near the middle of the village; his property passed to Hari Ram, a small shopkeeper living in the middle of the village. Nothing further occurred till the beginning of August when two persons were taken ill, Malan, dying on August 9th after eight days' fever, and her son Rana, who died on September 13th with a history of fever and enlarged glands, but also of syphilis. There is no definite evidence that these were plague cases, but rat mortality was also noticed at the beginning of

August, commencing in Hari Ram's cattle shed, which is close to Malan's house. In September, disease appeared among some Jats living near Malan's house and also among some Chamars occupying houses which adjoined, but had no direct communication with Malan's house and those occupied by the Jats, and there can be no doubt that these cases were bubonic plague although it was not until October when the disease, spreading more rapidly and frightening the people, was reported and so came to light. Interval 3 months. The subsequent epidemic 1897–98 was due to the spread from this focus by importation alone, recrudescence being, of course, absolutely excluded, and it is extremely important to notice that out of the total number of villages infected, 86, infection was traced in 72, or 84 per cent. Compare this with the 50 per cent. of the present investigation, where importation was a possibility, and in only half of which it was definitely traced. It was largely owing to the fact that, in districts which had previously been infected, it was difficult in many cases to account for the infection, as compared with those districts which were infected for the first time, that it was recognised that some other factor than importation was at work, and recrudescence began to be suspected. In the summary of the work of the Plague Commission, published by the Government of India, it is stated that a careful study of a Punjab district has shown that a history of possible importation could be obtained in 80 per cent. of the villages which became infected. In the case of a district previously uninfected, where the spread can only have been caused by importation, this is perfectly true, but in the absence of such qualification this statement is misleading, as the figures I have given show. The last cases of the epidemic of 1897-98 occurred in separate localities on June 3rd, 4th, 19th, 20th, and 23rd and July 24th. The first case of the next season occurred about the middle of September in a previously uninfected village, and of the first eight villages attacked, the source of infection was traced definitely in one only, very indefinitely in two, and none was discovered in the other five; these latter had not been infected before but it is significant that neighbouring villages had been attacked in the previous spring. The last cases of the 1898-99 epidemic took place on June 7th, June 23rd and August 15th. The first case of the next season which occurred on October 8th is interesting.

CASE 42. Village Sahiba, Garhshankar tahsil, Hoshiarpur district, population 1,595. In April, 1899, infection was imported by clothing from an infected village and there were four cases between April 12th and 19th, the epidemic being effectually cut short by evacuation. The next case occurred on October 8th, and no evidence of importation could be obtained. On this case Major (then Captain) C. H. James makes the following remarks in his report on the epidemic 1899-1900:—"In the case of Sahiba the question that at once arises "in the mind is, how did the disease get here? Had it remained dormant since "the previous spring and then again become active as the weather became cooler, "or had it been introduced from another village? If the latter, where had it come "from? We could get no evidence of the disease being imported. Indeed, there "were no villages infected for two months except Karnanan and this was at least "15 miles away. The nearest villages to Sahiba, which had been infected since "its previous attack, were Jaipur and Dial. But both these had been declared "free, the former on July 1st and the latter on August 12th, and moreover have "both since remained free of the disease. We are, therefore, forced to the con- "clusion that the infection had existed all along in Sahiba, in some form or other, "since the previous April."



MAP A.

SHOWING PLACES WHERE PLAGUE INFECTION WAS PRESENT THROUGHOUT THE QUIESCENT PERIOD AND THE SPREAD OF INFECTION THEREFROM BY IMPORTATION, 1 AUG - 15 DEC. 1911.

PROVINCIAL BOUNDARY. --------

RAILWAYS ——o——

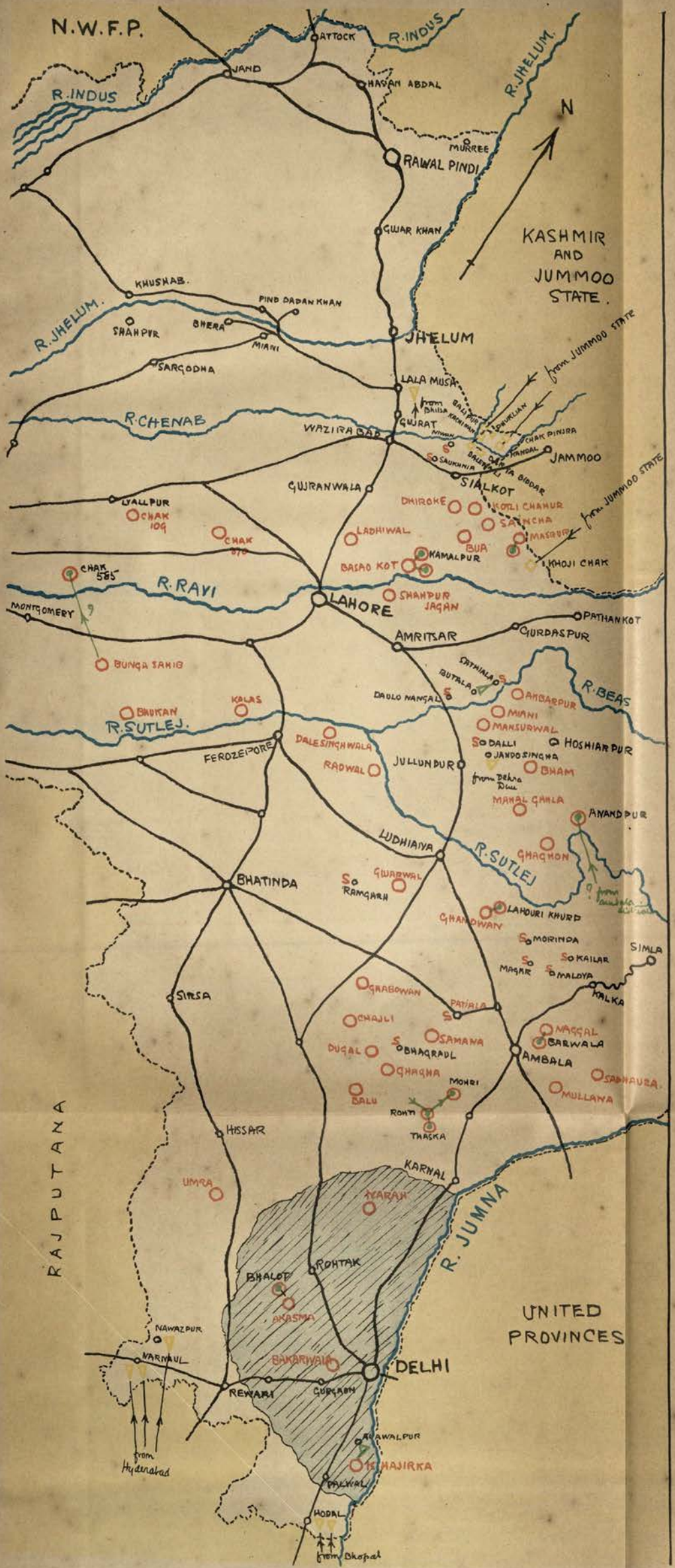
RIVERS ~~~~~

○ PLACES SHOWING INFECTION THROUGHOUT THE SUMMER

△ IMPORTED CASES NOT FOLLOWED BY EPIDEMIC

◎ IMPORTATION FOLLOWED BY EPIDEMIC

SCALE. 1 INCH = 32 MILES.



MAP B.

SHOWING PLACES WHERE EPIDEMIC PLAGUE REAPPEARED WITH NO EVIDENCE OF IMPORTATION TO ACCOUNT FOR THE INFECTION, AND THE SPREAD OF INFECTION THEREFROM, 1 AUG - 15 DEC. 1911.

PROVINCIAL BOUNDARY. --------

RAILWAYS. —o—o—

RIVERS. ~~~~~

AREA UNDER SPECIAL OBSERVATION.

○ REAPPEARANCE, NOT ACCOUNTED FOR BY IMPORTATION

△ IMPORTED CASES NOT FOLLOWED BY EPIDEMIC

◎ IMPORTATION FOLLOWED BY EPIDEMIC

S. SPORADIC CASES, WITH NO EPIDEMIC OR SUBSEQUENT INFECTION

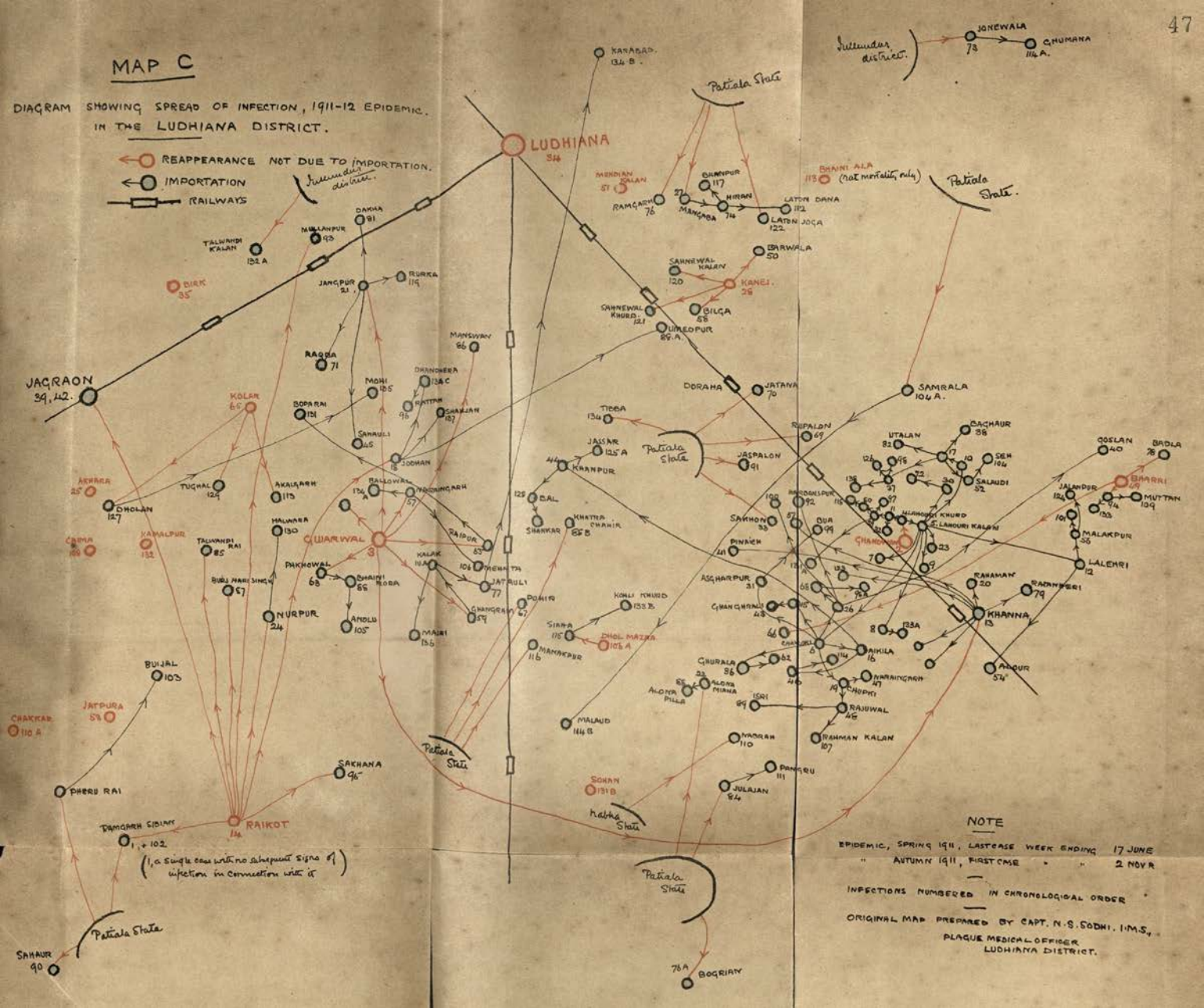
△ IMPORTED CASES FROM OUTSIDE PROVINCE

□ EPIDEMICS DUE TO IMPORTATION FROM OUTSIDE

SCALE. 1 INCH = 32 miles

MAP C

DIAGRAM SHOWING SPREAD OF INFECTION, 1911-12 EPIDEMIC. IN THE LUDHIANA DISTRICT.



NOTE

EPIDEMIC, SPRING 1911, LASTCASE WEEK ENDING 17 JUNE
" AUTUMN 1911, FIRSTCASE " " 2 NOVR

INFECTIONS NUMBERED IN CHRONOLOGICAL ORDER

ORIGINAL MAP PREPARED BY CAPT. N.S. SODHI, I.M.S., PLAGUE MEDICAL OFFICER LUDHIANA DISTRICT.

THE IMPORTANCE OF THE PERSISTENCE OF PLAGUE INFECTION IN CERTAIN VILLAGES DURING THE OFF-SEASON.

BY

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MEMBER, PLAGUE RESEARCH COMMISSION.

Plague infection may recur from year to year in a particular district or area in the following circumstances :—

Causes of repeated epidemics in a district.

Firstly.—Annual epidemics of plague in a district may be entirely dependant on re-importation from outside at the commencement of each plague season.

Secondly.—Infection may, year after year, persist during the off-season in one particular portion of that district in which conditions influencing plague are comparatively favourable during each off-season.

Thirdly.—Considering that an epidemic of plague in a town or village is often brought to a close largely by a diminution in the number of susceptible rats in that place, plague may persist in *any* town or village when it happens to become infected late in the plague season. In this case there might not be sufficient time for the diminution in the number of rats to take place, necessary to bring that epidemic to a close before the off-season sets in.

Material for study.

With a view to study the behaviour of plague in the villages of Poona district, and especially with a view to ascertain in what manner and in what particular localities, if any, plague persists during the off-season, the Plague Progress Reports of the Government of Bombay were analysed. In these reports the number of plague attacks and deaths are given for each village in the Presidency from 1899 to 1904.

Sources of error in material.

In reviewing these statistics, it must be pointed out at the outset that they are open to many fallacies, for example, these plague cases have in the majority of instances never been seen either during their illness or after death by a qualified medical practitioner; in many instances, especially when sporadic cases or deaths have been reported, a mistaken diagnosis may have been made. The symptoms and sudden death accompanying this disease are however so distinctive and so familiar to almost every inhabitant in this part of India, that we may safely assume that when a number of plague cases and deaths are reported to have occurred in an individual village, week after week and month after month, these cases and deaths have in most cases been correctly diagnosed as bubonic plague.

Again, it must be remembered that when a few isolated cases occur, a considerable responsibility rests on the individual who makes the diagnosis, and reports the case as plague. Should the diagnosis afterwards be suspected to be at fault, through the subsequent course of events in the village, or for other reasons, the individual, who reported a case of plague, might make himself very unpopular in that village owing to the inconvenience and alarm to which the inhabitants had been subjected. We may take it, therefore, that, especially in isolated cases occurring early in an epidemic

many plague cases and deaths are never reported as such, the death being ascribed to one of the more common causes such as fever, pneumonia, diarrhœa, &c., in fact anything but plague, until these deaths become more numerous and it is no longer possible to hide the presence of an epidemic in that village.

The material at our disposal in these reports is, however, so large that regarding circumstances in a broad light, a considerable amount of useful information with regard to the epidemiology of plague in villages may be gained by a study of the figures given in these reports.

Definition of "Plague" and "Off-Plague" seasons, Poona district.

We must now define what we mean by the "Plague" and "Off-Plague" seasons in Poona district. It has been shown in one of the reports of the Plague Commission published in the Journal of Hygiene, Vol. 10, No. 3, that conditions become rapidly favourable to plague (a fall in the temperature and a rise in the humidity accompanied by a rapid rise in the prevalence of rat fleas) during the month of June, and these conditions become rather more gradually unfavourable to plague in the month of March. The plague season of Poona city and presumably also that o surrounding district, where meteorological and other conditions vary but slightly, may therefore be said to last for nine months, *viz.*, from the beginning of July to the end of March. The off-season lasts approximately for the remaining three months, *viz.*, from the beginning of April to the end of June. These two seasons are clearly demonstrated in Chart No. VII, Journal of Hygiene, Vol. 10, No. 3.

Contrast between "Plague" and "Off-plague" seasons.

The contrast between the conditions (already detailed) unfavourable to plague during these three months and those favourable to plague during the first three months of the plague season is very great; so much so that if it be possible for rat plague to smoulder on in a village during the off-season months, it would undoubtedly at once manifest its presence in the form of human deaths during the early months of the plague season.

Villages which probably "carry over."

Regarding April, May and June, then, as the off-season for plague in Poona district, it follows that if all villages were collected in each year, which show plague cases and deaths during any of the first three months of the plague season (July, August or September) as well as in any of the six months which precede them, then these villages would include most of those that carried plague over this off-season period in each year. In some cases owing to the difficulties already mentioned, and our inability to discriminate between imported and local cases, the evidence of "carrying over" may be obscure; in others, however, as we shall see, it will be quite patent. It would be of great interest to see what particular characteristics are common to these villages which probably "carry over," and by observing their previous and subsequent plague histories, to see if they differ in any way from the more normal villages of the district.

Method of recording villages which "carry over."

In order to obtain a convenient and complete statistical, as well as a more or less graphic record of the plague history of all such villages, the following method was devised. In each year those villages in Poona district which apparently "carry over" (that is, show plague cases or deaths in July, August or September as well as in any of the six months preceding) were taken in order and their monthly plague cases and deaths for each of these nine months were entered on what we might describe as a "chart." This was done for each of the six years, 1899—1904, for which these records are available. The complete plague statistical history of these villages was then similarly traced for each remaining month of this 6-year period. The result is seen in Chart No. 1.

Imperfections of method.

It should clearly be understood that this method is not perfect. If it were, then there certainly would have been included a few more villages which, owing to the shortcomings in the system of reporting plague already mentioned, have failed to return cases and deaths during the off-season and the three months preceding it and so have not come into this category. On the other hand, it is possible that one or two of the villages in Chart No. 1 only appear to have carried over, plague may have died out naturally at the end of the season and infection may have been re-introduced early the following season from other villages in the district.

Small proportion of villages which "carry over."

It is also important to bear in mind that the villages which come into this category, form but a very small fraction of all the infected villages in Poona district during each of these six years; for instance, during the month of February 1902, 52 villages were infected in Poona district, whereas 13 places are given as infected in this chart and only 4 remained infected during the off-season. Again in March 1903, 87 places were infected, yet only 11 are mentioned in the chart and in only 4 did plague survive the subsequent off-season.

In tables Nos. 1, 2 and 3 the total number of infected villages during each month of the years 1902-1909 are given for Poona and its two adjacent districts Ahmednagar and Sholapur. These are villages which reported one or more plague deaths during each month. It will be seen that the number of villages infected during the off-season months form a very small proportion of all those infected during the months of the plague season. It should, therefore, not be impossible to deal prophylactically with these few villages infected during each off-season.

Examination of villages which carry over.

We will now examine Chart No. 1 in detail. We will exclude for the time being the various portions of Poona, *viz.*, Poona city, the Suburban area, Cantonments and Kirkee and will consider these subsequently.

With the exception then, of Poona; in the year 1899 at least three villages, as far as we can tell, carried plague over the off-season, *viz.*, Khed, Parbatti and Vadgaon. The remaining 5 which returned cases both in the first three months of the plague season as well as in the 6 months which preceded them, *viz.*, Lohagaon, Bhamburda, Baneri, Yerroda and Kadus are, according to the chart, all doubtful or only partially "bridged" the off-season. In 1900, Diwali alone probably carried over the off-season, and in 1901, Talegaon undoubtedly acted as a "bridge." In the year 1902, the off-season was probably bridged by Charoli, Lonavla, Saswad and Ottur; in 1903, wholly by Narayengaon, Katfal, Undawadi and Malthan and partially by Mekhali. In 1904, by Bavda, Nimbgaon-Ketki, Hardapsar and Mandavgaon.

Villages representative of all types in district.

Exclusive of Poona, therefore, 17 distinct villages as far as we can tell bridged over the off-season periods in these six years, and it will at once be observed by referring to a map of the district that these 17 villages are representative of every type and of every portion of Poona district. They vary in size from a population of under 400 to over 6,000. Some are situated on railways or main roads or near a large city, others are in a remote corner of the district. Some are situated in the higher portions of the district while others are in the lowest, some are on rivers, others at a distance from them.

Other features of interest.

Certain other striking features also emerge, *viz.*, unless a village bridge over the off-season or other exceptional influences come into play, it is comparatively rare for that village to be infected in two successive years, in other words, plague usually

dies out in each village at the end of each epidemic. Again, no individual village in this district acted as a bridge in more than one year. This shows that no single village in Poona district can be regarded as an endemic area of plague. Moreover, all these villages which either wholly or partially carried over plague during the off-season period have a normal seasonal variation of plague conditions, for when infected early, their plague season corresponds approximately with that of the district taken as a whole, or with that of Poona city (see Chart No. VII, Journal of Hygiene, Vol. 10, No. 3); as examples we may take the Charoli epidemic in 1899 and the Talegaon epidemics in 1899, 1902 and 1903.

Feature common to all villages that "carry over."

What peculiarities then are common to all these 17 villages? There is certainly one. All these villages apparently became infected towards the end of the plague season preceding the particular off-season which each bridged over. *An importation of infection late in the plague season then appears to favour the persistence of plague during the subsequent off-season in any village in Poona district in which such late importation chances to occur, but the infection dies out after each early epidemic*, for when in other years, these same villages chance to become infected early in the plague season, a normal epidemic results which in due course burns itself out completely before the subsequent off-season is reached.

Apparent exceptions to rule.

To this general rule there are, however, some instances which superficially appear to be exceptions. A reference to Chart No. 1 will show that Parbatti and Bhamburda were both infected in each of the six years. Although often infected early in the plague season, they appear at first sight to carry the infection over the intervening off-seasons. How do we know that in these places we have not endemic centres of plague? The question is regarded in a somewhat different light when we know that both these small villages are contiguous to Poona city, and indeed Bhamburda is now included within its Municipal limits. It is far more reasonable to suppose that these places also adhered to the general rule, *viz.*, that infection dies out after each epidemic and is re-introduced (in this case from Poona city) during the following plague seasons. Moreover, had plague infection been present in these two places during the intervening off-seasons, the epidemics would, in most cases, have made themselves manifest within the first three months of plague season for the reasons already quoted.

All these remarks would apply as well to Hardapsar which was also infected in six successive seasons. Hardapsar is a large village situated on the outskirts of Poona on the main road between that place and Sholapur. This village did actually carry over the off-season of 1904, after becoming infected probably in February, just before the end of that plague season. By contrasting the 1904 epidemic with those of previous years, this village gives a good example of the severe outbreak which usually follows during the first three months of the plague season when infection persists through the off-season and to which phenomenon we have already referred.

Two more apparent exceptions remain to be considered, *viz.*, Talegaon and Lonavla. These two places were also infected during six successive plague seasons. At first sight, it might be suggested that these constitute two endemic centres for plague in Poona district; indeed as has already been shown Talegaon in 1901 and Lonavla in 1902 undoubtedly did carry plague over these respective off-seasons. When, however, it is remembered that these two villages are both situated on the main road and railway between Bombay and Poona, it is far more reasonable to suppose that in each of the remaining epidemics, infection was freshly imported from one or other of these centres.

It will be seen from this that the only villages which are apparent exceptions to the rule laid down, are practically the only villages mentioned in the chart whose situation renders them exceptionally liable to repeated importation of infection from without.

Poona city may be a real exception.

Poona city with its various suburbs (including Kirkee) now remains to be considered. It is an exceptionally large town having an extensive trade with Bombay and the surrounding district, and hence can scarcely be compared with the much smaller villages of the district. It is difficult to learn from the chart, to what extent plague has carried over the off-seasons; infection was certainly carried over in 1899 and 1904, and in the case Kirkee in 1903, but it is impossible to state definitely if plague was carried over in any of the other years or not. Owing to its size, epidemics of Poona are more prolonged. Even if they commence fairly early in the plague season they may not have time to burn out before the following off-season is reached. For this reason Poona is more likely to act as an endemic plague centre than the average small village in the district.

Poona city may also be an apparent exception.

Even if infection do not continue through that off-season, it is—again owing to its size and therefore large trade communications—more liable to become re-infected early in the following plague season than the average village in the district. It might thus only appear to carry over a season when in reality it does not do so. Whatever may have happened in former years, in later years at any rate infection certainly appears inclined to die out in Poona city during the off-season.

Effect of immunity of rats.

This failure to carry over may be accounted for by an increasing immunity to plague of its rat population, which immunity has been shown to exist by experiments conducted by the Plague Research Commission. If this be the case, then, the importance of completely understanding the method by which plague bridges over the off-season in certain villages in the district will become greatly enhanced.

Comparison with Ahmednagar and Sholapur districts.

The plague seasons of the two districts adjoining Poona, namely, Ahmednagar and Sholapur, are similar to that of Poona. For comparison with those of the latter the village plague figures for these two districts have been tabulated in Chart Nos. 2 and 3 respectively. They exhibit similar characteristics.

There is one point in these two charts which is of exceptional interest and importance. In both districts, plague infection failed to carry over the off-season of 1900 and both these districts escaped the plague in the following season. Sholapur district was indeed not generally infected again till comparatively late in the plague season of 1902 and Ahmednagar not till 1903. On the other hand, in 1903, when many villages handed the infection over the off-season in both districts, a severe and widespread epidemic ensued.

The importance of persistence of infection in villages.

The persistence of plague infection in villages during the off-season is thus of considerable importance. Its importance would be further increased as the chance of plague carrying over in large towns diminishes, and especially if we were able to devise some practical scheme or system whereby we could detect in each year the few villages in the district which are likely to carry plague over the off-season, such as villages which happen to be infected late, or those in which either cases or deaths occur during the off-seasons. Having detected them these villages could be isolated while they yet remain infected, or we might anticipate nature by diminishing their rat population by some means other than plague. Even if we were thus able merely to delay the re-infection

of the district, each year, till the three most favourable months of the plague season is past, the severity of the district epidemic would be greatly diminished. With fewer villages infected during the plague season, there would probably be fewer still infected during the following off-season. The fewer there are then infected the more effectually could these be dealt with; there might indeed be no necessity again for such prophylactic measures during the following off-season.

Résum of items of interest.

On reviewing the information derived from studying the charts, the following interesting points suggest themselves with regard to the villages of Poona district.

1. Villages which have a large trade communication with infected places, which are on the main road or railway, or which are adjacent to large plague infected centres, are liable to become infected frequently.

2. Villages which are remotely situated in the district usually escape plague for several years in succession.

3. The recurrence of annual epidemics of plague in the district is not necessarily dependent on yearly importations from endemic or other centres of plague outside.

4. There is no single village or group of villages in the district where plague is endemic.

5. Whenever villages are infected early in the plague season, infection dies out completely either before or when the following off-season is reached.

6. When infection is implanted late, however, it occasionally continues over the following off-season.

7. The explanation of this is that the epizootic has not sufficient time to exhaust the rat population of that village before the off-season commences, and it is by this method that infection is bridged over the off-season in Poona district.

8. When in one year no village carried plague over the off-season in two adjacent districts, these entire districts remained practically free from local infection for one or two years afterwards, thus showing that importation of infection from without is of minor importance, when compared with the persistence of infection within.

Comparison between Poona and West Coast districts.

There is one more question that it might be of interest to raise. In Poona district as we have seen plague is carried over the off-season with some difficulty, and for fairly long intervals its towns and villages have been free from the disease. Yet in the towns of the West Coast which have never suffered as severely from plague epidemics as those of Poona district, infection appears to persist through the off-season with much greater facility—Bombay, Ratnagiri and Mangalore have seldom been free from plague and it might almost be said to be endemic in some of these places. What is the explanation of this?

Regarding the question in the light of what we have said about Poona district, the probable explanation of this phenomenon is that in the West Coast towns there is not the same contrast between favourable conditions in the plague season and unfavourable conditions in the off-season. The climate is more equable and meteorological conditions do not vary much, hence there is also less seasonal variation in the flea prevalence in these towns. In consequence of this epidemics of plague are never very severe and the rat population is probably not reduced, during an epizootic, to the same extent that it is by an epizootic in Poona city by the time the off-season

arrives. This fact together with a comparatively high flea prevalence during the hot weather would readily explain the persistence of plague in West Coast towns during the off-season.

Before concluding this paper I must acknowledge my indebtedness and gratitude to Dr. G. D. Chitre, late of the Plague Research Commission, and to Mr. Pansare of the Bombay Bacteriological Laboratory for the valuable assistance they have afforded me in compiling these statistics.

Conclusions.

I should finally like to emphasise the most important conclusions that can be deduced from this work, namely :—

1. There is no single and constant endemic centre for plague in Poona district, nor are the annual epidemics of plague entirely dependent on importation from outside the district.

2. Plague may persist during the off-season in *any* village in Poona district and such persistence is favoured chiefly by the introduction of infection into that village late in the preceding plague season.

3. Those villages which "bridge over" the off-season in Poona district form but a very small fraction of the total number of villages infected in that district during the previous plague season.

Table No. 1.

Table showing the number of infected villages in Poona District 1902-1909. Population 995,330. Towns 11. Villages 1,178. Total 1,189.

Months.	Poona District.									
	1902.	1903.	1904.	1905.	1906.	1907.	1908.	1909.	Monthly Total.	Monthly Average.
January ...	...	77	61	33	4	37	16	40	268	38·29
February ...	...	91	57	24	4	17	17	28	238	34·00
March ...	...	87	44	16	2	11	13	40	213	30·43
April ...	14	29	28	6	2	6	9	3	97	12·12
May ...	1	7	8	1	1	4	2	3	27	3·37
June ...	1	1	3	0	1	0	1	1	8	1·00
July ...	3	2	6	0	1	2	4	0	18	2·25
August ...	8	6	13	0	7	4	7	5	50	6·25
September .	18	16	20	4	35	9	28	10	140	17·50
October ...	41	31	29	5	81	14	46	14	261	32·62
November .	36	40	36	13	94	23	48	19	309	38·60
December .	50	59	43	13	64	22	39	14	304	38·00

TABLE No. 2.

Table showing the number of infected villages in Ahmednagar District 1902-1909. Population 837,695. Towns 8. Villages 1.341. Total 1,349.

Months.	Ahmednagar District.									
	1902.	1903.	1904.	1905.	1906.	1907.	1908.	1909.	Monthly Total.	Monthly Average.
January ...	.....	34	129	59	3	2	0	1	228	32·57
February ...	.....	203	124	47	4	2	0	2	382	54·57
March ...	.....	100	128	45	5	0	0	2	280	40·00
April ...	0	67	77	24	3	0	0	2	173	21·62
May ...	0	17	10	8	1	0	0	0	36	4·50
June ...	0	4	0	0	0	0	0	0	4	0·50
July ...	0	11	8	1	0	0	0	0	20	2·50
August ...	0	45	23	3	2	0	0	0	73	9·12
September ..	0	105	55	3	3	1	2	0	169	21·12
October ...	2	177	83	7	7	1	4	0	281	35·12
November ...	5	199	76	6	5	1	4	...	296	42·29
December ...	7	148	61	3	3	0	2	...	224	32·00

TABLE No. 3.

Table showing the number of infected villages in Sholapur District 1902-1909. Population 720,977. Towns 7. Villages 712. Total 719.

Months.	Sholapur District.									
	1902.	1903.	1904.	1905.	1906.	1907.	1908.	1909.	Monthly Total.	Monthly Average.
January	.....	86	179	31	3	0	5	9	313	44·71
February	.....	104	182	28	2	0	4	5	325	46·43
March	.....	90	152	25	3	1	3	4	278	39·71
April	9	46	68	11	4	1	0	0	139	17·37
May	3	10	18	4	0	1	0	0	36	4·50
June	0	2	3	0	0	0	0	0	5	0·62
July	4	15	10	3	0	1	0	0	33	4·12
August	6	37	15	5	0	1	1	0	65	8·12
September	12	77	24	6	1	1	3	0	124	15·50
October	12	142	35	6	0	1	4	0	200	25·00
November	21	170	38	5	1	6	6	...	247	35·29
December	54	176	34	4	0	5	10	...	283	40·43

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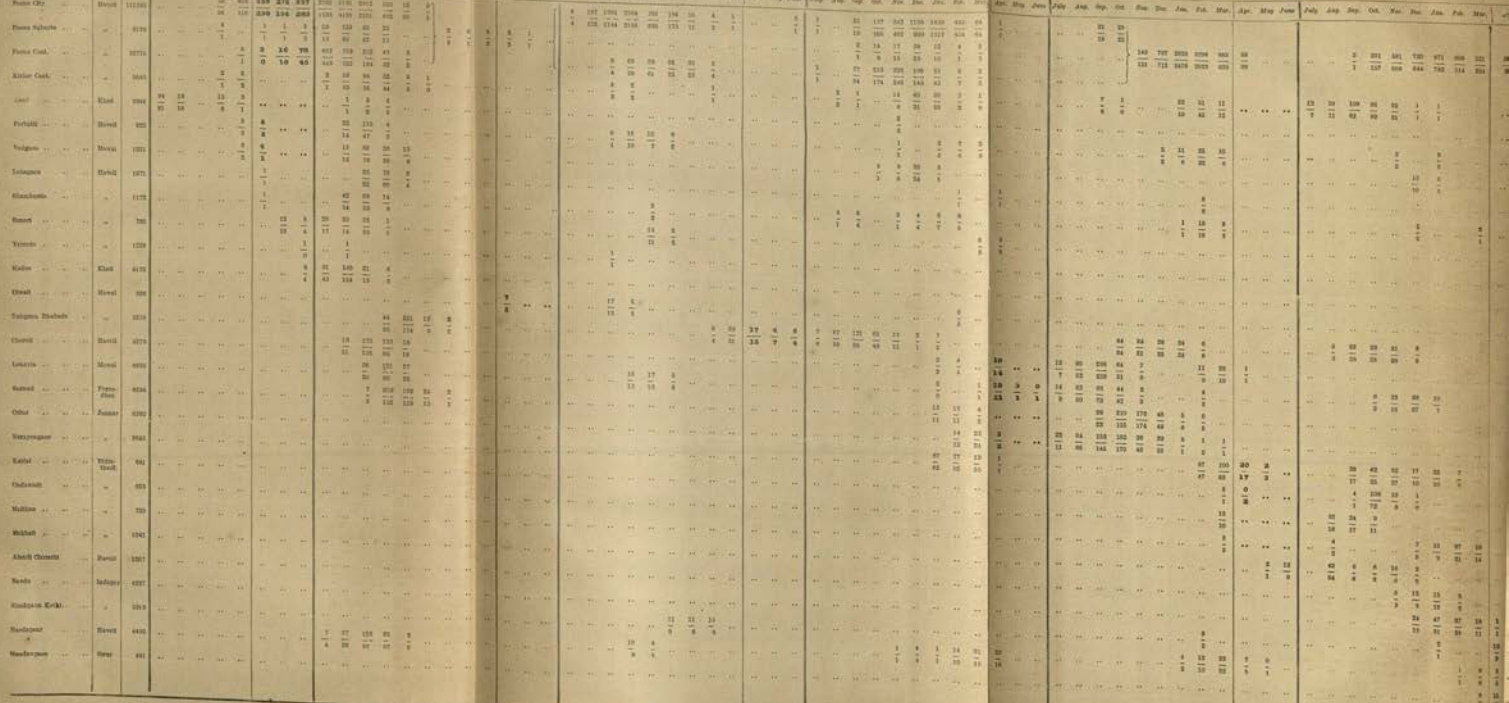
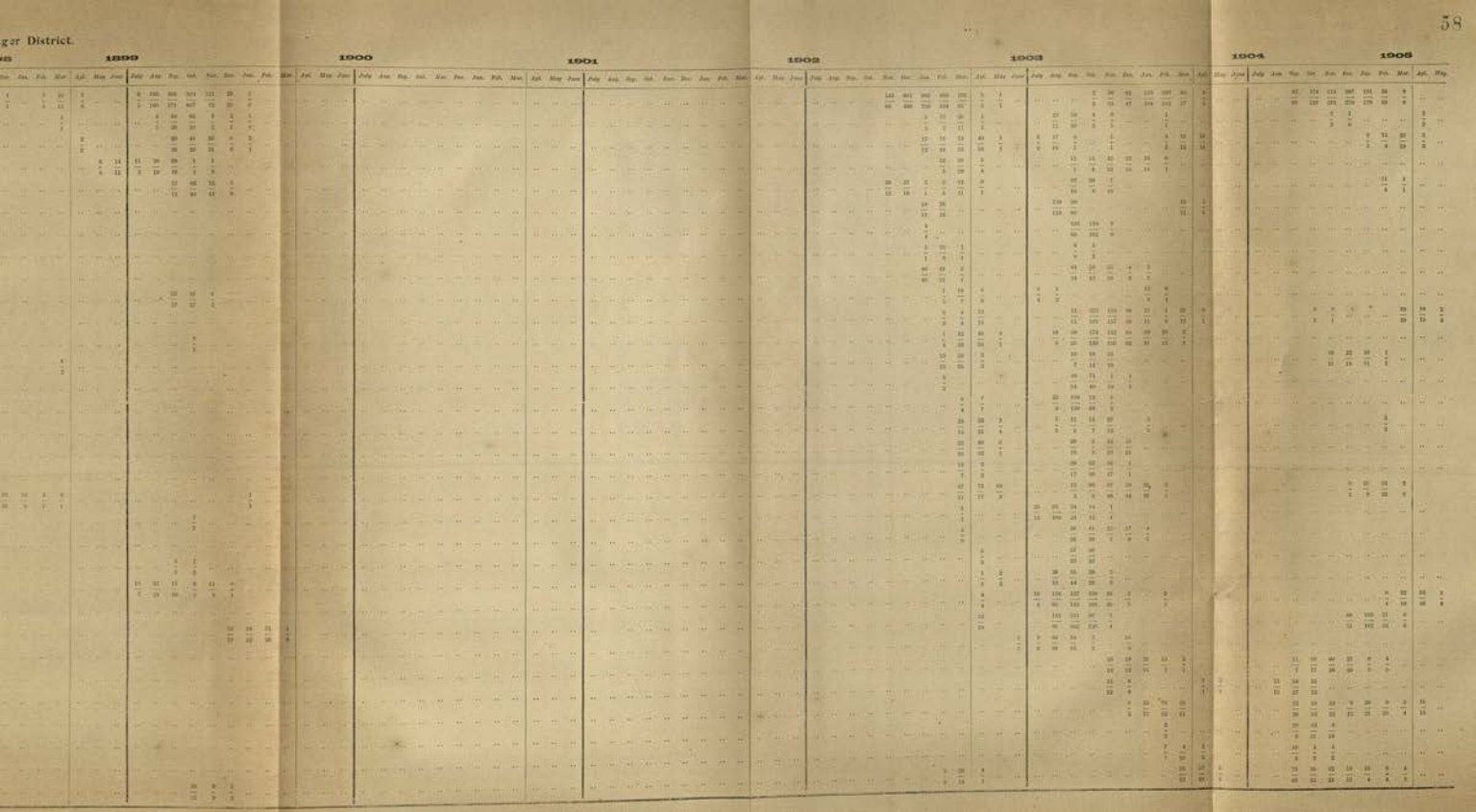
a District.

1898 1899 1900 1901 1902 1903 1904 1905

CHART No. I. Poona District.



ger District.



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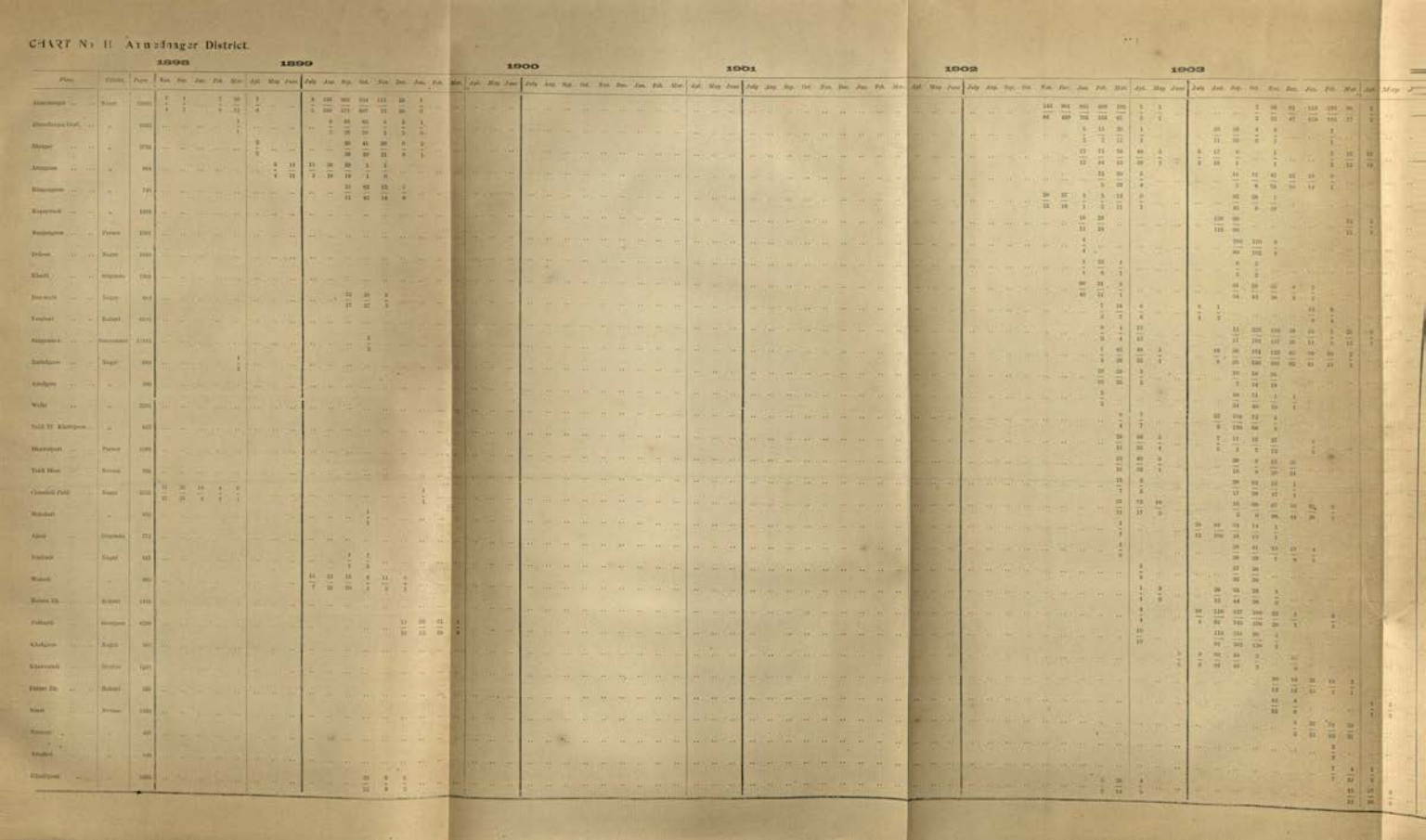

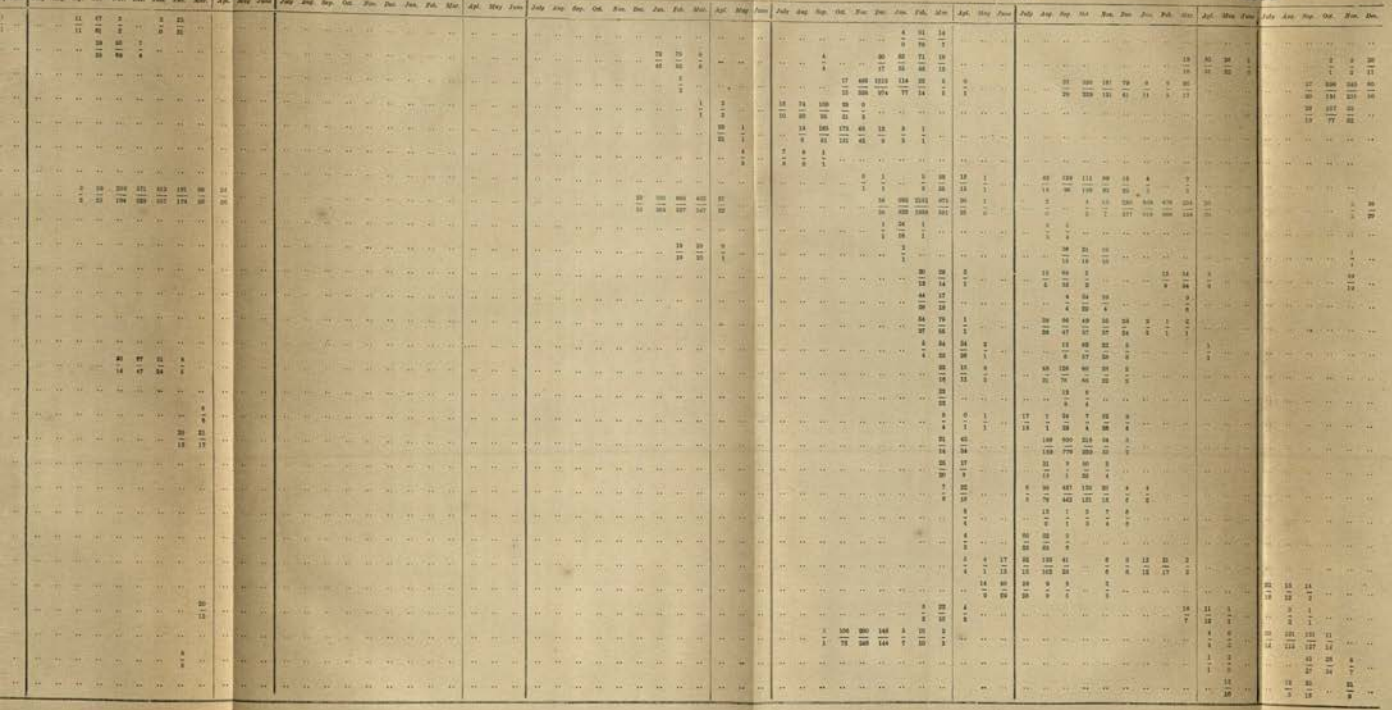
1899
1900
1901
1902
1903
1904

CHART No. III. Sholapur District.

Place.	Taluka.	Popn.	1899															1900																1901															
			Jan.	Feb.	Mar.	Apl.	May	June	July	Aug.	Sep.	Oct.	Nov.	Dec.	Jan.	Feb.	Mar.	Apl.	May	June	July	Aug.	Sep.	Oct.	Nov.	Dec.	Jan.	Feb.	Mar.	Apl.	May	June	July	Aug.	Sep.	Oct.	Nov.	Dec.	Jan.	Feb.	Mar.	Apl.	May	June	July	Aug.	Sep.	Oct.	Nov.	Dec.	Jan.	Feb.	Mar.
Kandalgaon	Sholapur	1887	..	..	17/8	13/14	3/3	..	..	..	11/11	67/61	2/2	..	3/0	25/21	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Boramani	„	2803	..	..	..	..	..	..	..	..	..	39/25	93/69	7/4	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	72/42	75/52	5/8
Barsi	Barsi	24242	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3/2	..
Achegaon	Sholapur	1513	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1/1
Mangrul	„	2246	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Togarhalli	„	426	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Madha	Madha	5309	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Sholapur	Sholapur	77521	..	..	..	..	..	..	..	..	3/2	39/25	209/194	571/528	413/397	181/174	96/83	24/26	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	20/19	393/264	698/527	432/347
Pappnas	Madha	211	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Tirth	Sholapur	766	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	19/10	10/10
Puluj	Pandharpur.	1843	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Ghoti	Karmala	672	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Lawool	Madha	1768	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Rophal or Ropic	Karmala	707	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Alerwadi	Sholapur	2091	..	..	..	..	..	..	..	..	..	..	30/14	67/47	31/24	8/5	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Dharphal	„	1527	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Bohalli	Pandharpur.	859	..	..	..	..	..	..	..	..	..	..	..	..	..	..	6/6	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Karkambe	„	5570	..	..	..	..	..	..	..	..	..	..	..	..	..	20/13	21/17	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Nerle	Karmala	257	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Sangola	Sangola	4763	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Telgaon	Sholapur	419	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Dahiwali	Karmala	519	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Tulsi	Madha	861	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Khardi	Pandharpur.	1549	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Deogaon	Sholapur	1536	..	..	..	..	..	..	..	..	..	..	..	..	..	..	20/18	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Wahang	„	4452	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Aurad	„	1464	..	..	..	..	..	..	..	..	..	..	..	..	..	8/8	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Mahaloong	Malsiras	904	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..

DEFECTIVE REGISTRATION OF PLAGUE DEATHS, AN EXPLANATION FOR THE REAPPEARANCE OF PLAGUE WITHOUT EVIDENCE OF IMPORTATION OF INFECTION.

BY

DR. D. A. TURKHUD, M.B., C.M. (Edin.).

The present paper is based on the results of a special investigation which was undertaken in Satara district of the Bombay Presidency from 1908—1910 to inquire into the origin of the recurring epidemics of plague in the Valva Taluq of that district.

A preliminary tour through the taluq was first made, in the months of March, April and May 1908 to obtain a general idea of the nature of the district, the condition of the villages, the habits of the people, and any other special circumstances likely to favour plague. Thirty-six villages in the taluq were visited, and inquiries were made by consulting the village records to ascertain if plague had prevailed in them during the previous year 1907. The only records available for this purpose were, (1) the Register of Births and Deaths, which is kept in every village, and (2) the Plague Register, which is started in a village when it becomes plague infected, and which is a record of all plague attacks and deaths from day to day. The inquiries showed that in 35 out of the 36 villages plague was more or less prevalent during the year 1907. Inquiries were also made to ascertain, if possible, how plague started in these villages. These inquiries showed that the first death registered as plague was not, as a matter of fact, the first death from plague which had occurred in the village. Plague, as a rule, had been prevailing in the village some time previous to the date on which the first case had been reported, the unrecognised plague deaths were registered as deaths from fever, diarrhœa, asthma or some other cause, and evidence of the presence of plague could generally be obtained by finding an increase in the number of deaths from all causes in the village. A plan was devised to detect this increased death-rate as follows :—

It was ascertained that the normal death-rate per mille per annum of a village in this district was about 25, the population taken into account in calculating this death-rate being that of the Census of 1901. The following formula was used to obtain the normal number of deaths per month :—

$$\frac{\text{(Population of village according to Census of 1901)} \times 25}{1{,}000 \times 12}$$

Any rise in the number of deaths above the figure obtained by the formula, therefore, raised suspicion as to the presence of an epidemic. Careful inquiries were then made among the relatives of the deceased, and also among their neighbours ; with a little tact, the actual cause of death, if due to plague. was soon found out.

The following example will show how this was done. One of the first villages visited was Kande, population 1,551. According to the village registes an outbreak of plague started in the village on the 28th of September 1907 with two plague deaths : (1) Lakhoo Rama Patil, and (2) Ganoo bin Mahardoo Patil. Inquiries showed that Lakhoo had not gone out of the village, while Ganoo had visited a village called Shitur in Kolhapur State, but the village was not infected at the time of his visit. These two deaths, therefore, gave no clue to the source of infection, and had the inquiry ended here the epidemic might have been regarded as a recrudescence of the previous infection in the village.

But an examination of the Death Register showed the following mortality :—

Month.	Plague deaths.	Deaths from all causes.
1907.		
January	12	13
February	22	22
March	13	13
April	.....	3
May	.....	3
June	.....	1
July	.....	2
August	.....	5
September	6	16
October	22	22
November	8	11
December	18	18

According to the formula given above, the average normal monthly mortality of the village should be 3. As a matter of fact the number of deaths in the month of August had risen to 5 and in September to 16, although in the latter month 6 deaths were returned as plague. A close examination of the Death Register showed that a number of deaths which occurred in August and September were registered as due to fever, and that these deaths had for the most part occurred among a class of people known as " Vaddars," a wandering tribe of earth and stone workers who had come to the village from a place called Nerle which was at the time infected with plague. The first case among these people to arouse suspicion was registered on the 20th of August, and it was found on visiting the spot where this death occurred that two persons had died in the same house a fortnight later, *viz.*, on 4th and 5th September. The Vaddars soon after left the village. The villagers themselves admitted, when questioned on the matter, that these were true cases of plague, yet in the register they had been ascribed to ' fever. ' It was further found that another suspicious death had occurred on the 3rd September—a woman, a potter's wife, died in a house near that occupied by the " Vaddars." In the next house two other deaths occurred among potters on the 9th and 10th September. These deaths also were recorded in the register as due to fever; but the village potters admitted at the time of my inquiry that these persons had died of plague with buboes. Two other persons who had dealings with the Vaddars also died of suspicious plague, one on the 16th and the other on the 17th September. But the village officers did not declare the village plague-infected till the 28th of September, *i.e.*, 40 days after the death of the first plague case.

Incidentally attention may be drawn to the fact that the number of deaths recorded as due to plague at the close of the epidemic in January, February and March was in excess of the real number of deaths, which in all probability could be attributed to that disease. All deaths, for example, in the months of February and March were returned as due to plague, while only one in January is recorded as due to other causes than plague.

Name of village.	Population.	Date of the first plague death as recorded in the village registers.	Dates of the actual first death from plague, and also subsequent plague deaths, which occurred before the village was officially declared plague-infected, and the causes to which these deaths were attributed.
Kande	1551	28th September 1907	20th August 1907 Fever. 3rd September 1907 ,, 4th ,, ,, ,, 5th ,, ,, ,, 9th ,, ,, ,, 10th ,, ,, ,, 16th ,, ,, ,, 17th ,, ,, ,,
Sagaon	2504	2nd September 1907	16th July 1907 Fever. 20th August ,, ,,
Aitwade Khurd	2516	13th September 1907	1st July 1907 Diarrhœa. 3rd ,, ,, Fever. 6th ,, ,, ,, 8th ,, ,, Diarrhœa. 9th August ,, Fever. 10th ,, ,, Diarrhœa. 11th ,, ,, Fever. 26th ,, ,, ,, 27th ,, ,, ,,
Mangle	4760	10th September 1907	15th August 1907 Fever. 29th ,, ,, ,, 6th September 1907 ,, 10th ,, ,, ,,
Chikurde	4039	12th September 1907	? June 1907 Fever. 6th July ,, ,, 10th ,, ,, ,, 18th ,, ,, ,, 1st September ,, Diarrhœa. 2nd ,, ,, Fever.
Karanjwade	1309	25th May 1907	11th April 1907 Fever. Two other suspicious cases, dates not entered in notes.
Aitwade Budruk	3538	22nd August 1907	7th July 1907 Fever. 21st ,, ,, ,, ? ,, ,, ,, 30th ,, ,, ,,
Kurlap	1866	14th September 1907	5th August 1907 Diarrhœa. ? ,, ,, ,,
Washi	1290	28th February 1907 (1st epidemic). 8th September 1907 (2nd epidemic).	27th January 1907 Fever. 27th ,, ,, ,, 3rd August ,, ,, 8th ,, ,, ,,
Yellur	2991	20th June 1907	14th June 1907 Fever. 15th ,, ,, ,,
Bahadurwadi ...	2156	21st July 1907	In April 1907, 3 suspicious deaths from fever. In May 1907, 4 suspicious deaths from fever. 24th June 1907 Fever. ? July ,, ,,
Kanegaon	811	21st October 1907	17th September 1907 Fever. Between 17th September and 21st October six suspicious plague deaths occurred, all attributed to other causes.
Tandulwadi	1570	9th October 1907	2nd June 1907 Fever. 30th ,, ,, ,, 10th July ,, ,, In August 8 suspicious deaths among residents from Islampur who had gone to live at Tandulwadi owing to plague at Islampur. In September, 3 deaths among residents from Islampur, and 4 among those from Bahadurwadi; all attributed to causes other than plague.
Bagni	5642	9th March 1907	1st March 1907 Fever. 6th ,, ,, Diarrhœa.
Sawalwadi	768	5th October 1907	23rd September 1907 Fever. 30th ,, ,, ,,
Peth	6808	18th January 1907	8th January 1907 Fever. 12th ,, ,, ,,
Kameri	5074	9th April 1907	25th February 1907 Fever. 16th March ,, ,, 27th ,, ,, ,, 30th ,, ,, ,, 1st April ,, ,,
Kapuskhed	2126	5th August 1907...	14th July 1907 Fever. 17th ,, ,, ,, 26th ,, ,, ,,
Takari	1339	17th July 1907	16th June 1907 Fever. 20th ,, ,, ,, 23rd ,, ,, ,, 5th July ,, ,,
Yede Machindra	1602	24th August 1907	13th July 1907 Fever.
Nerle	7504	12th May 1907	9th April 1907 Fever. 12th ,, ,, ,, 21st ,, ,, ,, 23rd ,, ,, ,, 3rd May ,, ,, 8th ,, ,, ,,

The accompanying table gives a list of some of the villages in which on inquiry it was found that the first cases of plague reported in the village registers were not actually the first plague cases. The list is not a complete one, but it shows clearly that the village registers are neither accurate nor reliable. Any attempt made therefore to trace the origin of the infection in villages from the first cases of plague recorded in the village registers must necessarily prove futile and lead to quite erroneous conclusions.

Various reasons can be advanced to account for the incorrect registration of deaths. In some cases the cause of death, especially if no buboes are developed, is not recognised by the unskilled villagers and death is reported as due to fever. No attempt is made by the village officers, who unfortunately are not trained medical men, but who are nevertheless responsible for the correctness of the register, to verify the cause of death unless some crime is suspected. Sometimes, even when the village officers are aware that a death is due to plague, they will purposely record it under another cause. The reason for this deliberate suppression of facts was found to be this : in Satara district there is a rule that whenever the first plague case occurs in a village a special report must be made by the village officers daily for the fortnight following. The report has to be forwarded to the Taluq headquarters, which may be some miles distant, by a special messenger. In order to avoid the inconvenience of sending a special messenger so far, the men concerned do their best to ignore all early deaths from plague, and make every effort to postpone notifying the presence of plague in their village by registering the deaths as due to fever, diarrhœa or asthma. When, however, the number of deaths increase rapidly there is no alternative left but to attribute the mortality to plague. The presence of plague is then notified to higher authorities.

On account of the unreliability of the village registers, it was thought necessary to employ a special agency of our own to verify the causes of deaths in the villages in our area. This agency was also utilised to collect early information about any mortality among rats, to find out the first cases of human plague, and to trace the origin of infection, if possible.

For this purpose the selected area was divided into three portions, each consisting of a dozen villages. An Inspector was appointed to look after each portion or 'circle.' Subsequently a fourth circle was added to the area to keep under observation certain villages to the east of the Taluq.

The Inspectors were carefully instructed as regards their duties. They had to visit all the villages in their respective circles regularly once a week and in a certain definite order. On visiting a village the Inspector was required to examine the village Death Register and personally inquire into the cause of every death which had occurred there during the week subsequent to his previous visit. He had to make special inquiries about the deaths which appeared to be suspiciously like plague, and ascertain if there was any mortality among the village rats. After making these inquiries he had to submit his report on a special Diary Form. A separate form was used for each village. The Inspector had instructions to send the forms by post to the Special Medical Officer on the last day of the week ; but if he heard any rumours of dead rats or thought that a certain villager's death was due to plague, or detected an actual plague case, the diary form had to be posted at once with a special report on the result of his inquiries to trace the origin of infection. In this manner continuous and authentic records were obtained showing the cause of every death in every village under observation. Suspicious deaths were thereby detected early, and it was thus possible to trace the origin of plague epidemics.

The information obtained from these diary forms was found to be sufficient to enable the Special Medical Officer to keep a close watch on the

mortality of the villages under observation ; they showed whether deaths had occurred in the same house or locality, whether in any particular community or among children. It was thus possible to suspect early, even at the headquarters, the presence of an epidemic in a village and to direct the Inspectors to make special inquiries in the case of suspicious deaths which had escaped their notice. The work of the Inspectors, however, had to be watched very strictly by paying surprise visits to the villages, for they used all kinds of devices to evade their work. They sometimes, instead of visiting villages, sent for the registers from a convenient place in the neighbourhood and copied therefrom the necessary information to fill in their diary forms ; at other times they did not visit a village, but reported that the village register was not available and that consequently they could not submit the weekly return. A Special Supervising Inspector had to be appointed, and it was quite possible to exercise a proper check over the work by means of surprise visits, by examining the Death Registers, and by asking the Inspectors to point out the houses where recent deaths had occurred.

The number of villages kept under special observation in this manner in the four circles was as follows :—

Karad	Circle,	15	villages.
Shirala	„	13	„
Islampur	„	13	„
Tasgaon	„	13	„
Total ...		54	

A detailed account of the observations carried on in these villages will, it is hoped, be published elsewhere at a subsequent date, but it may be stated here in short that in every instance where a village became infected by plague, the source of infection could be traced to another infected village, and that no outbreak could be attributed to recrudescence or recurrence without importation of infection.

The observations carried on in the Tasgaon circle showed that plague persisted in that locality from August 1908 without cessation in the off-season, and was still prevalent there at the close of the year 1910. The chart given below gives a good idea as to how these villages went on infecting and re-infecting each other and thus maintained the infection continuously during two years 1909 and 1910.

MONTHS.	Bhilawdi.	Akalkhop.	Ammapur.	Burli.	Nagthane.	Palus.	Dhavli.	Yelavi.	Turchi.	Tasgaon.
August 1908 ...	+	...	...	...	...	...	...	...	...	...
September ...	+	...	...	..	...	+	...	...	...	...
October ...	+	+	...	...	...	+	...	...	...	...
November ...	+	+	...	...	...	+	...	...	...	...
December ...	+	+	...	...	...	+	...	...	...	...
January 1909 ...	+	+	...	...	...	+	...	...	...	...
February ...	+	+	...	+	...	+	...	...	...	...
March ...	...	+	...	+	...	+	...	...	...	...
April ...	...	...	...	+	...	...	...	...	...	...
May ...	...	...	+	...	...	...	...	...	...	...
June ...	...	...	+	...	...	...	...	...	...	...
July ...	...	...	+	...	+	...	...	...	...	...
August ...	+	...	+	+	+	...	...	...	...	...
September ...	+	+	+	+	...	+	...	...	...	...
October ...	+	+	+	+	+	+	...	...	...	...
November ...	+	+	...	+	+	+	...	...	...	...
December ...	+	+	..	+	+	+	...	...	...	...
January 1910 ..	+	+	...	+	+	+	...	...	...	...
February ...	+	...	...	+	...	+	+	...	...	...
March ...	+	...	...	+	...	+	+	...	...	...
April ...	...	...	...	+	...	...	+	...	...	...
May ...	...	...	...	...	...	...	+	...	...	...
June ...	...	...	...	...	...	...	+	...	...	...
July ...	...	...	...	...	...	...	+	+	...	...
August ...	...	...	...	...	...	...	+	+	...	...
September ...	...	...	...	...	...	...	+	+	+	+
October ...	...	...	...	...	..	...	+	+	+	+
November ...	...	...	...	...	...	...	...	+	+	+
December ...	...	...	...	...	...	...	...	...	...	+

It is interesting to note that during the quiescent plague period, in the month of May, in this circle, only one village—and that a different one each year—was infected ; this village was the source from which all other villages in the circle became infected in each epidemic. Our investigations in the other three circles also showed that every outbreak of plague which occurred in the villages could be traced to the importation of infection from another infected village. It is thus quite evident that erroneous deductions may be made as to the origin of an outbreak if the first cases of plague which are officially registered are taken as the starting point of that outbreak ; accurate conclusions can only be arrived at if the actual first cases of plague are carefully ascertained after detailed inquiry on the spot.

The facts already recorded sufficiently justify this conclusion, yet it may be worth while recording the story of an outbreak of plague in the village of Virmade which, as far as the official records showed, was free from plague, but which nevertheless was smitten by the disease late in the epidemic season; the disease persisted throughout the quiescent plague period and this village was the focus from which the surrounding district became infected in the following epidemic season. The presence of this epidemic came to the notice of the Collector in July and he asked me to make a special inquiry as to its origin.

Virmade is a small village—population 768—about ten miles to the north of Satara town ; plague was not known to be present in this part of the district during the year 1909, but inquiries, which were instituted on the Collector's orders, showed that the people in the village had been suffering from plague from the month of April. The death register of the village showed that six deaths had been recorded in that month and that four of these had occurred in one household at about the same time ; these deaths had been registered as due to fever, but investigation proved that every one of them was due to plague, the infection having been introduced into the village from Bombay. In the month of May dead rats were still being found in the village, but no deaths were recorded. In June two suspicious deaths from fever were reported from the same part of the village. In July there were as many as thirty-eight deaths, but the presence of plague in the village was not notified by the village officers till the 28th of that month. No plague register was kept nor were the entries in the death register accurate because the village officers were among the first to run away from the village and live elsewhere. The villagers followed their example and migrated into the neighbouring villages in the Taluqs of Satara, Wai, and Jaoli. Some of the inhabitants of Virmade who had left that village while actually suffering from plague were traced to the different villages to which they had fled and were found to have died there. Inquiries showed that every one of these deaths was entered in the village death registers as due to fever and subsequently all these villages were infected with plague.

The outbreak of plague in this village showed in a striking manner the apathy generally exhibited by the village officers in notifying the presence of plague especially during the off-season ; it demonstrated how a solitary infected village became the starting point of a widespread epidemic and how the first plague cases and deaths in infected villages are not officially recognised.

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ALL-INDIA SANITARY CONFERENCE—MADRAS—NOVEMBER 1912.

A SIMPLE METHOD OF RAT DESTRUCTION.

BY

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Chief Plague Medical Officer, Punjab.

The gases produced by burning sulphur in air have long been used as a disinfectant, and in recent years for the destruction of rats. The Clayton apparatus for the production of these gases, SO_2 and SO_3, in large quantities and their propulsion is now largely used for the destruction of rats on board ship; experiments however with this apparatus in Indian houses have not proved successful, principally because penetration of the gases into the rat burrows and nests, where the rodent and its parasites live, was not sufficient; moreover the apparatus is elaborate and expensive and on these grounds quite out of the question for general use.

Sirdar Dayal Singh Man, Sirdar Bahadur, the President of the Council of Regency of the Faridkot State, during the course of some experiments he was carrying out with sulphur with the object of destroying weevils in grain, conceived the idea that rats might be readily destroyed by introducing sulphur fumes directly into the rat hole and he devised a simple and cheap stove for the purpose. It is simply an ordinary small "angethi" closed at the top with a lid from which proceeds a tube that can be inserted directly into the rat hole; a little ignited charcoal, or "bhusa" is placed in the stove and on the top of this a drachm or so of sulphur on a piece of tin; the end of the tube is then inserted into the rat hole, the space between the outside of the tube and the edges of the hole being closed with clay, and air is blown in through the stove into the rat hole for about five minutes by a cheap pair of bellows fitted into a small hole at the bottom of the stove; all holes communicating with the original one, indicated by the issue of smoke, are immediately closed with clay and, when the tube is withdrawn, the original hole is similarly stopped; the fact that rats are killed in their holes and that the bodies remain there must be a strong deterrent to reoccupation. The stove costs Rs. 3, and can be obtained from the Superintendent of the Model Farm, Faridkot. Sirdar Dayal Singh proceeded to try his method on the town of Faridkot; at the time of my visit there 900 houses had been treated, 2,899 rat holes being stopped; two months afterwards only eight of these were found reopened and only twelve new ones had appeared. I saw the stove operated on some field rat burrows, and actually saw that rats were killed in burrows at a considerable distance from the hole by the process.

The methods seemed so simple and satisfactory that it was decided to carry it out in a Punjab town and Sargodha was selected for the experiment. Sargodha is the headquarter town of the Jhelum Canal Colony and a big grain collecting and distributing centre, with a population of 8,849 and some 1,700 houses including a large "Mandi" or market with big godowns and shops; plague appears here annually in the spring and there is a fairly general exodus of people at this time which dislocates trade and helps to spread plague far and wide. The experiment was carried out very thoroughly by Captain Southon, I.M.S., and the idea seemed to appeal to the people, probably because the smoking out of rats is not unknown to them, an ordinary "ghurra" with a hole in the bottom and burning "bhusa" being used for the purpose. Plague unfortunately began before operations were started, and this complicated matters considerably; many dead rats were found, and four of the coolies employed contracted plague. The Dayal Singh stove did not prove altogether satisfactory for these extensive operations, as the bellows was not powerful enough to completely penetrate the

very large holes found in this town, built principally of burnt brick in mud plaster and Captain Southon substituted an ordinary large earthenware "ghurra", for the stove, and used a country goat skin bellows which is much more powerful than the one supplied with the stove; later as it was found that the "ghurra" cracked after some time from the continued heat, an empty petrol drum was used.

The great difficulty in the process is the clearing of houses and shops which is absolutely necessary if the rat holes are to be got at; it speaks well for the co-operation of the people in this instance, that the clearing of some of the large shops and godowns, costing the occupier some thirty or forty rupees, was thoroughly done. Lanterns are absolutely necessary for finding the holes in dark rooms, and a close watch has to be kept in adjoining rooms and houses when holes are being smoked, as other exits may exist there which require immediate closing to prevent the escape of the rats.

The following is a summary of the work done:—

	No.
Total number of houses	1,689
Ditto rooms	6,500
Ditto rat holes	9,583
Number of working days	40
Cost, about	Rs. 1,000
Average number of stoves employed	8
Average number of coolies employed	23

The principal item of expenditure was labour, very dear in this part of the world, which amounted to over six hundred rupees, the materials for smoking only cost eighty rupees.

Three months after the operations very few rats were present as shown by trapping and according to the towns people. The campaign will be repeated in January when a better idea will be obtained of the success of the process, judging from the number of rat holes found then, and there are good grounds for supposing that by this means, the spring epidemic will be prevented. The process itself is exceedingly simple, appeals to the people, especially with regard to the depredations of the field rat, and is very inexpensive, particularly if "bhusa" smoke which Sirdar Dayal Singh now claims to be all that is necessary, alone is used. We hope to repeat this experiment in certain other selected places during the coming winter, and we propose teaching the method all over the province.

Three months after the operations in Sargodha, the grain market was persistently and carefully trapped for three weeks with 28 traps, the total catch being 64 rats; before the operations with the same number of traps, this number of rats would have been accounted for in a single day. It is evident that a very great reduction in the rat population was caused by the operations and that recovery is by no means rapid.

A small village with 84 rooms has recently been done in three days, very few holes have been re-opened.

GRAIN AND THE GRAIN TRADE CONSIDERED AS FACTORS IN THE PERSISTENCE AND DISSEMINATION OF PLAGUE IN INDIA

BY

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There are few now who deny the importance of the role played by the rat in the spread of bubonic plague. The association of rats and grain stores is a fact familiar to all. Is it not a little remarkable therefore that grain should have received so little consideration, as a factor in the dissemination and persistence of plague infection, at the hands of Sanitarians and Epidemiologists in India as it has done? Grain (and all other forms of merchandise) are allowed to pass throughout the country without let or hindrance. In Madras and certain of the Native States where the traveller from plague-stricken areas is subjected to varying degrees of surveillance, grain is allowed to enter unrestricted and treated as if it were above suspicion. This is all the more surprising when one studies the numerous and voluminous plague reports that have been produced in India during the last fifteen years. Instances in which plague amongst rats was first noted in granaries, markets and such like, are encountered on all sides. We have no time in this short resume of a large subject to do more than touch on some of these instances.

It will serve a useful purpose if we pause for a moment to consider the possible reasons for this neglect of grain and the grain trade as important etiological factors of plague.

In the very beginning of the present Indian pandemic suspicion fell on grain for it was in the large grain warehouses of the Bombay docks that the disease first declared itself and from which it emanated. Little was then known about the epidemiology of the disease and it was emphasized by the earlier workers, that, had grain been the infecting agent a sudden widespread epidemic would have resulted. Hankin did some experiments which showed that grain was little likely to be the infecting agent, and Bannerman* in 1906 in a paper on the Spread of Plague sums up the then available evidence as follows, "We may safely conclude therefore that grain cannot be incriminated."

It is from another point of view that the writer now wishes to incriminate grain, *viz.*, as a vehicle second to none in importance for the transmission of infected rats and rat fleas from place to place. In the last interim report of the Advisory Committee for Plague Investigation in India† it is stated that "Plague appears to be commonly imported into a fresh locality about the persons of human beings though the transference of infected rats and fleas in merchandise must be considered." It appears to me that this statement emphasizes unduly the importance of the human factor and puts less stress on merchandise than the facts warrant.

It is usually difficult to obtain satisfying evidence as to the source of infection when investigating an outbreak of plague. If one is content with a

* The Spread of Plague in India—Journal of Hygiene, Vol. VI, No. 2, 1906.
† Journal of Hygiene, Vol. X, No. 3, 1910.

history of a recent arrival from a plague-infected place such is generally forthcoming in infected and non-infected towns alike in these days of railway communication. If it is early in the plague season, the possibility of the infection being a survival of last year's epidemic has always to be borne in mind.

One has more hope of arriving at the truth when investigating the origin of an outbreak in a place infected for the first time than in one that has been the scene of annual visitations.

Last year (1911) Hyderabad-Deccan was infected for the first time in its history. Infection was stated to have been brought in with grain.

In 1911 Vaniyambadi in the Salem District was infected after plague-free interval of eight years. Infection was ascribed to grain.

In 1909 Palghat in the Coimbatore District suffered from its first epidemic and infection here too was almost certainly introduced with grain.

In 1911 Java* suffered from its first outbreak. Rice was responsible for the introduction of infection.

In 1905 I obtained two plague-infected *Mus Rattus* in a grain godown in the Malakand Fort. The grain had just been imported from down country. There was no plague, at that time, in the North-West Frontier Province.

The history of the introduction of plague into the United Provinces I shall treat of at some length. Grain seems there too to have been largely responsible.

Such instances might be multiplied to almost any extent. The above are sufficient to indicate the frequency with which plague infection has been ascribed to grain. That it is probably the most important of all infecting agents will appear from what follows. It is even possible that certain areas owe their immunity from plague to their independence of outside supplies of grain.

Plague in the United Provinces.

In this paper I shall deal chiefly with plague in the United Provinces for it is this portion of India that has claimed most of my time and attention as a Member of the Plague Research Commission during the last two years. The early history of plague in this Province is illustrative of what commonly occurs and is worthy attention. Plague spread from Calcutta to the Western Districts of Bengal in the cold weather of 1898-99. The Bengal District of Saran was very severely attacked. The danger that the adjacent United Province district of Ballia ran was at once fully recognised, as very free communication exists between portions of the Ballia District and the towns of Chapra and Revalganj in Saran. Nevertheless it was not till January 1901 that Ballia became infected and it has hardly ever been plague-free since. Plague refugees flocked to the Ballia District from Bengal; as many as 11,500 are reported as having arrived in the months of November, December and January 1900-1901. Ballia's first case occurred in Raniganj and it is surely not without significance that it occurred in a house immediately adjoining the shop of a grain dealer who had extensive dealings in grain with Revelganj in Saran then severely infected. The part that grain appeared to play was apparent to the then Collector and Civil Surgeon who attributed the rapidity of the spread of the disease from Raniganj to the fact that this town was a large grain mart supplying numerous surrounding villages.

* The first Plague Epidemic in the Dutch Indies by Dr. J. J. Van Loghem Extrait du "Janus," Leyde 1912.

Plague also broke out simultaneously in the City of Benares. It is not so clear whence infection was brought, but the reports emphasize the fact that the classes trading in grain, spices and other food stuffs were particularly affected.

July and August were plague-free but the following season 1901-02 the disease reappeared and this year again the Benares division with the adjoining district of Allahabad was responsible for practically all the plague of the Province. An inquiry into the grain trade of these eastern districts for this year is of interest. At this time Bengal was the only imminent source of danger to the United Provinces. We find that the United Provinces imported by rail and river during the year ending 31st March 1902, 1,910,882 maunds of grain from Bengal and of this 1,824,507 maunds went to the Benares Division* and of the small balance 45,784 maunds went to Allahabad.

Of the total grains and pulses imported into the United Provinces this year from all other provinces, *viz.*, 5,100,000 maunds, 2,300,000 went to the Benares Division.

Of the 1,824,507 maunds of Bengal grain imported into the Benares Division 150,073 maunds were wheat and the remainder chiefly rice, a kharif crop. The excessive grain importation was due to the unfavourable harvest gathered in the eastern districts of the United Provinces in 1900-1901.

From this year onward the Provinces have never been quite free from the disease. It is the eastern end that has suffered most, chiefly because climatic or other conditions there prevailing are never sufficiently adverse to cause the complete disappearance of the disease in the off season.

In the year 1902-03 we again find the severity of the disease in the different trade blocks bearing a close relationship to the amount of grain imported from other infected areas.

In subsequent years the relationship was not so close, it was complicated by the varying degrees with which the different parts of the Province carried over plague infection through the off seasons. This phenomenon is comparatively infrequent however in the west of the Province. Here if we take the imports of grain into each trade block for a representative year (from the other trade blocks of the United Provinces, as well as from other Provinces) and compare them with the amount of plague that that area has suffered from in the past, some interesting and significant facts are elicited. I have taken 1910-11 Trade returns as the latest available at the time of writing. This was a year of rather more than average prosperity in which the surplus of grain in the Province as a whole was somewhat greater than can be expected to occur. The imports of grain from outside the Province would certainly not be in excess of those of a normal year.

Trade blocks in the West of the United Provinces.	Plague death-rate per mille for all epidemics up to June 1911.	Imports of grain per head of population in 1910-11 from other Provinces as well as from other blocks of these Provinces.
Meerut Division (excluding the plague free district of Dehra Dun)	51·5	22·3
Agra Division	43·8	15·2
Rohilkhand Division	25	5·5
Districts of Gonda, Bahraich, Sitapur and Kheri.	5·7	1·4

* The Benares Division in the Trade Returns prior to 1908 included the present Benares and Gorakkpur Divisions.

I have expressed the imports of grain as amounts per head of population, as this seems to me the most convenient method of describing the respective dependence of each area on imported food stuffs.

I do not suggest any degree of exact correlation between these two sets of figures. There are such numerous other factors of importance having a direct bearing on the question that it would be foolish to try to do so. All the same, they are to my mind extremely suggestive.

Bundelkhand Division of the United Provinces.

The four districts of the United Provinces that lie south of the river Jumna on the northern edge of the Central Indian plateau have enjoyed a comparative immunity from plague, that is most striking. This phenomenon is one of those that claimed especial attention from the Members of the Plague Commission who were in charge of the observations in the United Provinces. Banda, the easternmost district of the four, was the one selected for detailed observations. A complete year's work was done in Banda, the head-quarters of the district, and revealed somewhat unlooked for results. These may be summarised as follows :—

(1) Mus rattus is extraordinarily numerous in Banda.

(2) X. cheopis, the common rat flea, occurs on the rats in greater abundance here than anywhere else in the United Provinces where similar observations have been made.

(3) The rats are remarkably susceptible to plague.

(4) Though the density of rural population is low, 193 persons to the square mile, it is not so low as that of some Punjab districts that have suffered very severely from plague.

(5) The human intercourse by rail and road with infected areas though less than in some other parts of the Province is by no means so restricted as to explain the immunity (if the human being be looked upon as a common carrier of plague infection). In some years many plague refugees are reported to have found an asylum here.

(6) The year in which our observations were made was not one in which meteorological conditions departed appreciably from the normal.

(7) The meteorological conditions are very similar to those pertaining in some other places that have been severely infected.

(8) No measures at all are taken to keep plague out.

An inquiry into the trade of the district revealed the fact that from the point of view of grain the district is very largely self-supporting. No figures representative of the trade of the district are available in the Annual Reports of Inland Trade, but the Great Indian Peninsula Railway have compiled for me a statement showing the amount of grain and pulse received at all the Railway stations and out receiving stations of the district for each month of the year 1910-11. This shows that in spite of the sparsity of the population, the total year's imports of grain amounted to only 2·9 seers per head of population (total imports 46·794 maunds). Small as this figure is, it is larger than I was led to expect from local inquiry. I have not been able to ascertain the chief sources of Banda's supplies.

Banda town exports considerable quantities of grain, but imports practically none.

I believe Banda's immunity to plague to be chiefly accounted for by the smallness of its grain imports. Should infection succeed in gaining a foothold early in the plague season (*i.e.*, in the post-monsoon months) a very severe outbreak would almost certainly result.

The influence of Grain Stores and Markets on the persistence of plague infection.

In 1908, when studying the seasonal prevalence of plague in Belgaum, I was struck by the fact that during the epidemic not only were rats much more plentiful in the market and grain godowns than elsewhere, but a much higher percentage of these rats were found to be plague-infected than of those caught in the remaining infected areas. I called attention in my report* to the danger of the weekly market (so common in small towns in all parts of India) and the danger of the common cutcha grain godown. Subsequent experience has much strengthened these opinions and I now regard the methods of storing grain in India as largely responsible for the persistence of plague in this country. I have no time to more than touch on the facts that have led me to this belief, but as an example of the influence that large accumulations of grain may have on the plague history of a town, I should like to cite the case of Cawnpore. As most of you are aware Cawnpore occupies a unique position as a trading centre amongst the inland towns of India. As a collecting and distributing centre for grain it has no rival. During the year ending February 1912, 265,348 bullock cart loads of merchandise (most of it grain) entered the city; this is equivalent to about six million maunds. These imports are by road alone. By rail, during the year ending 31st March 1911, Cawnpore City imported 2,169,175 maunds of grain, pulse and oilseeds. Now the early plague history of Cawnpore city was almost as exceptional as is its position as a trade centre. Whereas epidemics of plague in the United Provinces reach their height in March or April (more commonly the latter month in the Western districts) Cawnpore's first epidemic reached its height in October, the next two epidemics in November, and the two following in December.

The first year in which Cawnpore had the "normal" type of epidemic was in 1907-08,† a year of great famine in which the kharif crop almost completely failed and Cawnpore's stores of grain must have been for a time reduced very much below normal.

Now the early type of epidemic, I believe, to have been due to the very large rat population (which Cawnpore still has), and this in its turn is due chiefly to the huge accumulations of grain. The excessive rat population enabled plague infection to survive the adverse conditions of the hot weather and to assume epidemic proportions before the other influencing factors had attained their optima.

There is only one other fact in the Plague History of this Provinces to which I shall refer.

Jhansi town has suffered from very few epidemics of plague. Jhansi imports its grain chiefly from Bhopal and Gwalior. I have not the plague figures for Gwalior, but it is interesting to note that Bhopal's epidemics 1902-03, 1903-04, 1904-05 and 1911-12 occurred in the same years as those in which Jhansi, too, suffered from epidemic plague and in no other years was Jhansi infected.

Famine and Plague.

I have endeavoured above to indicate in a general way that there appears to be a somewhat close relationship between imports of grain from infected areas and plague. At first sight this contention might be deemed inadmissible in face of the well-known fact that in years of scarcity and famine, when the imports of grain are larger than normal, plague is rarely, if ever, severe. This ceases to be anomalous when it is considered how little food there is for rats in times of famine. The rat population must of necessity be much below

* Journal of Hygiene, Vol. X, No. 3, 1910.

† In this year the imports of grain into the United Provinces exceeded the exports by 20,510,000 maunds of grain.

its normal standard*. This being so the harm that would accrue from the introduction of plague infection is much less than would obtain in normal years.

1905-06 and 1907-08 both years of famine in the United Provinces were years of very little plague.

Another fact of importance in the present plague problem is the extent to which grain is held up after harvest by traders and agriculturists waiting for a favourable market. This practice appears to be on the increase, in years of plenty, in certain parts of India, and the cultivator seems to becoming less of a hand to mouth individual than he formerly was.

The Storage of Grain.

Whilst engaged at work in the United Provinces, the Sanitary Commissioner of those Provinces was good enough to address for me a circular letter to the Collectors of all the districts of the Province asking for information under the following heads :—

(1) What is the usual method of storing grain in your district, in godowns, pits, etc.? Does the method vary in towns and villages and if so how ? Is grain stored loose or in gunny bags ?

(2) What claims are made by the owners of grain stores for the special suitability of the different methods in vogue ?

(3) If pits are not used are there any local features that render such a method of storing grain unsuited to the district, *e.g.*, dampness of soil, high-water level, etc. ?

(4) What are the chief trade centres for grain in the district, and are the godowns in these pucca or kutcha built ? If pits are built how are they constructed ?

(5) In years of plenty is grain for export commonly held up for long period in towns or villages waiting for a favourable market or is it common practice to export as soon after harvest as possible ?

(6) Have any attempts ever been made to construct " rat-proof " godowns and if so, with what success ?

(7) If grain pits are used, are they infested with rats ? If not, what is the usually accepted explanation of the fact, can any other explanation be offered ?

(8) Are there any local pecularities in the district as to the methods of storing domestic supplies of grain in private houses ?

(9) Have any facts been noted in connection with past epidemics of plague in your district which point to market or grain stores as having been commonly the starting point of such epidemics ? If you have personal experience of this in other than your present district, I should be glad of particulars.

From the replies received from the forty-eight districts much useful and interesting information has been collected.

There is no time now to give a detailed summary of these replies, the following facts, however, are among the most interesting that have come to light.

The most popular method of storing grain in bulk is loose in underground pits (khatties).

* It must also be remembered that years of famine are usually years of very deficient rainfall and low degree of humidity. Deficient humidity we have shown to be detrimental to the rat flea.

This method is usually preferred because of its

(1) low cost of construction;

(2) the freedom of grain so stored from attacks of rats and weevils;

(3) the length of time that grain so stored will keep good;

(4) in one district it was claimed by the Bunnias that the grain kept in pits absorbed moisture and so increased in weight, a most desirable result from their point of view.

The freedom of pits from rats is noted in all districts where pit-storing is common and is in nearly every case ascribed to the great heat developed in the interior of the pit.

Pits are not used:—

(1) Where the water-level is high.

(2) Where the soil is stony.

(3) Where the soil is salt and loose.

Extensive irrigation sometimes renders pit-storing of grain impracticable. It is interesting to note that pits are little if at all used in the eastern districts where plague survives the off season more readily than elsewhere. High water-level is chiefly responsible for this. Godown storing is preferred in a few places even where pits are feasible if there is a rapid turn over of the grain. Grain in gunny bags in godowns is more convenient to handle and weigh. There are only two pits to my knowledge in Cawnpore City.

Grain stored loose is nearly always surrounded with bhusa which is said to protect the grain from rats.

The experience and opinions of the district officers as to whether granaries and markets play an important part as the staring places of epidemics vary considerably and it is difficult to summarise the replies received. Twenty of them note the frequency with which epidemics start or persist in centres of the grain trade or in markets. Twenty-three have never noted such facts. Five of the districts having been practically plague-free no opinion is expressed on the point by their Collectors.

Some of the replies are emphatic. Thus the District Magistrate, Fatehpur wrote :—

> "The chief plague-infected locality in this district this year is a tract in the neighbourhood of Bindki, the principal grain mart of the district. An examination of the plague records since 1907 shows that plague usually makes its first appearance in Tahsil Khajuha (in which Bindki is situated) and that Tahsil usually bears the brunt of the attack. The connection of plague with grain stores seems to be established by the history of plague in this district."

Again, the Collector of Bulandshahr, says :—

> "Dihai and Khurja, the two places from which the recent years infection has spread over the district are the two most important centres of the grain trade and it was noted last year that the plague mortality was exceedingly high in all the places enumerated above as being grain centres."

Summary and Conclusions.

I have dealt with the subject only from an epidemiological standpoint, but I hope I have brought forward sufficient evidence to show that grain and the grain trade as factors in the spread of plague in an agricultural country such as this is worthy the careful consideration of this Conference.

It may be true that infected rat fleas are carried about in the luggage or on the persons of travellers, but there is no evidence to show that they are except over very short distances and in small numbers. The chances against a flea so carried finding its natural host would be very considerable. Moreover when one remembers the very considerable degree of immunity that the rats in the plague-stricken areas of India have now attained, one would expect to find the disease becoming less widely diffused year by year if human agency (using the term in its narrowest sense) were chiefly responsible for the spread of the disease. I am not aware that this is so. On the other hand rats and rat fleas introduced with grain go as a rule to the very portions of a town where rats are most numerous and their chances if they be infected, of setting light to an epidemic are very great indeed.

If grain stores could be kept free from rats "infection" could be introduced into them with impunity.

If my contention that grain is the chief vehicle for the spread of plague infection over distances be a correct one, the plague problem in India ceases to be the hopeless one that so many consider it to-day. Much could be done.

The building of pucca rat proof grain godowns especially in the larger grain distributing centres; the dissociation of large grain stores from dwelling houses: and the regulation of grain imports from plague-stricken areas are the three desiderata to which our plague policy should tend.

The disinfection of grain in bulk on railways should not present insuperable difficulties. Progress must of necessity be slow, but thus directed, I believe, success would eventually attend our efforts.

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PLAGUE IN THE MADRAS PRESIDENCY.

BY

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The remarkable immunity from plague of the greater part of the Madras Presidency as contrasted with the rest of India presents an interesting problem for investigation and it has long been a matter for speculation as to whether this immunity is due to any special climatic or other factor adverse to the spread of plague or is in any degree due to the special preventive measures adopted in this Presidency. The Plague Research Commission have now been engaged in an investigation into plague in Madras for over a year and it is proposed to put before the Conference in a brief form some of the main points as to the distribution of plague in the Presidency and the facts as to the relation of this distribution to geographical areas and to physical and climatic conditions. This paper will merely deal with some of the material collected, without any deductions being drawn, in order that the facts necessary to an appreciation of the problem to be considered may be before the Conference to form a basis of discussion. Our own personal conclusions will of course be published later.

The Madras Presidency exhibits a great variety in climate and other physical features. Extending as the Presidency does over 12 degrees of Northern latitude and presenting elevations from sea-level to 8,000 feet, the climate is found to vary greatly in different parts of it. If climate has any influence on the epidemiology of plague then we should find its effects demonstrated in comparatively small areas. As a matter of fact when we analyse the facts as to the distribution of plague we find that it is markedly limited to certain natural geographical regions and not to any special administrative districts. Also within the affected areas plague presents different epidemiological characteristics in different parts.

In investigating plague in the Madras Presidency we must also include Mysore State and consider the South of India as a whole. We can conveniently divide the Presidency and Mysore into 5 main divisions according to physical conditions, and the following is a short account of these divisions and of the incidence of plague in them :—

(1) The largest and most important of the divisions is the long and fairly broad area lying between the Eastern Ghauts and their continuation Southwards after their junction with the Western Ghauts in the Travancore Range, and the East Coast. This includes the whole of the East Coast Districts from Ganjam in the North to Tinevelly in the South and also includes Trichinopoly District. This extensive area is almost entirely low-lying and varies from sea-level to a few hundred feet above the sea. These districts have almost entirely escaped plague, the only outbreaks that have occurred being represented by under 200 deaths in the 13 years from 1898 to 1910 in Madras City, South Arcot and Trichinopoly District and by a moderate epidemic in the Palni and Dindigul Taluqs in Madura District which however occurred in higher-lying portions at an elevation of about 1,000 feet where this district adjoins Coimbatore.

(2) The second division is represented by the parts of the central district of North Arcot, Salem and Coimbatore which lie at a general elevation of about 1,000 feet above sea-level and slope up to the Mysore Plateau. Taking this area all over it has suffered in only a mild degree from plague. There have however been several outbreaks of considerable severity in some of the larger Municipalities, notably in Vaniambadi, Tiruputtur, Salem and Coimbatore. These outbreaks, while of great severity when they did occur, have been

infrequent and there have been long intervals in many cases before the recurrence of an epidemic.

(3) The third large geographical area is that lying between the Eastern and the Western Ghauts and is divided into two portions:—

(*a*) The more southerly part is the Mysore Plateau and includes Mysore State and the adjoining parts of the Madras Presidency, the Taluqs of Hosur and Kollegal. Its general elevation is about 3,000 feet above sea-level. This plateau is an endemic centre of plague, which has been continuously present since its first invasion in 1908. Even in such a small area as Hosur Taluq plague is practically endemic. Apart from the Nilgiri Hills, Hosur and Kolligal Taluqs are the coolest parts of the Presidency proper and these areas are the only parts of the Presidency in which plague tends to persist although the epidemics are not very severe.

(*b*) The plateau to the North of Mysore lies at a lower level, a general elevation of about 1,500 feet above the sea and includes Bellary and Anantapur Districts, and parts of Kurnool and Cuddapah Districts. Of these Districts, Bellary has suffered most severely and in fact the epidemics have been so severe in Bellary District in some years that it has accounted for nearly half the plague deaths in the Presidency. Cuddapah, the lowest-lying and hottest of the ceded Districts, has suffered least from plague. While Bellary District has had years of very severe epidemics, there have also been years in which it has almost entirely escaped plague. A comparison between plague in Mysore State and in Bellary District will show that in Mysore plague maintains a more constant level than in Bellary, but does not reach the same epidemic severity nor die out to the same extent. The result is that on an average of 13 years the incidence of plague per mille in these two areas is practically the same. The difference in the plague incidence in Bellary and the adjoining districts, Anantapur and Kurnool, cannot be explained on climatic grounds. The proximity of Bellary District to the plague-infected Dharwar District may afford a possible explanation of its liability to infection.

(4) The fourth area into which we have divided the Presidency for our purposes is the narrow strip lying between the Western Ghauts and the sea, and includes the districts of South Canara and Malabar. There has been extremely little plague in these districts except in the sea-ports of Mangalore, Tellicherry, Cannanore and Calicut. Mangalore has been continuously affected since the first introduction of plague in 1902, but after the first outbreak plague has persisted but has never been severe. The other three ports have had small limited outbreaks. Passing from North to South along the West Coast plague diminishes rapidly in amount until it ceases entirely in Cochin. The severity of plague in South Canara, Malabar and Cochin respectively vary, as 7; 1; 0.

(5) The fifth and last area to be described is the Nilgiris Plateau at a generel elevation of 6,500 feet to 7,500 feet. This area has a temperate rather than tropical climate, thus differing from the rest of the Presidency. Plague has been present each year since its first introduction in 1903 but the total number of deaths is not large. Calculated on its small and scattered population the incidence of plague per mille is high, in fact, next in order to Bellary District. Plague in this district is very persistent, but epidemics are never severe

and in this respect conform to the Mysore type but are even less severe.

A detailed analysis of the plague figures in the affected districts, distinguishing deaths in towns from those in rural areas, gives a better idea of plague distribution in certain taluqs. The figures of plague deaths in towns and rural areas separately are available for the years from 1901 to 1910 and these 10 years will be dealt with.

Table No. I shows that 28,477 deaths have occurred in town circles and 39,972 in rural areas. The proportions are:—

Plague deaths in rural areas	63·8	per cent.
„ „ Towns over 10,000 population...	30·9	„
„ „ 5,000 to 10,000 population ...	5·3	„

Of the deaths in rural areas 83·8 per cent. have occurred in Bellary, Salem and Coimbatore Districts.

Table No. II shows the average annual plague death-rate per mille in the towns and rural areas of the districts, and it will be seen that the only district which returns a high rural death-rate is Bellary. The plague death-rate per mille in Bellary district is :—

Rural areas	2·52
Town circles	6·59

Next in order of severity of rural death-rate, but much lower, is the high-lying cold Nilgiris District. Coimbatore and Salem Districts follow after the Nilgiris District and as will be shown later their rural mortality is to a considerable extent due to the high-lying taluqs of Kollegal and Hosur which are situated at an elevation of about 3,000 feet above sea-level.

The rural death rate in Anantapur is still lower and in the remaining districts is extremely low.

Excluding Bellary District the remaining districts which are infected return plague deaths as follows :—

Plague deaths in rural areas	20,113
„ „ „ towns over 10,000 population...	15,950
„ „ „ 5,000 to 10,000 population	1,237

Of the 17,189 deaths in town circles 13,237 occurred in the following 8 towns :—

Coimbatore District.—Coimbatore and Kollegal	3,483
Salem District.—Salem, Vaniambadi and Tirrupattur	5,943
North Arcot District.—Vellore and Ambar	1,200
South Canara.—Mangalore	2,611
Total ...	13,237

Of the deaths in rural areas Hosur Taluq in Salem District returned 3,417, and Kollegal Taluq in Coimbatore returned 2,785.

The distribution of plague in the districts other than Bellary is thus :—

In 8 large towns	13,237 or 35·5	per cent.
In other towns over 10,000 population... ...	2,713 or 7·3	„
In towns of 5,000 to 10,000 population ...	1,237 or 3·3	„
In rural areas of Hosur Taluq...	3,417 or 9·1	„
In rural areas of Kollegal Taluq	2,785 or 7·5	„
In other rural areas	13,911 or 37·3	„

The average annual plague death-rate per mille in these areas and in Bellary District is :—

In the 8 large towns (outside Bellary District)	4·82
In other towns over 10,000 population	·34
In towns of 5,000 to 10,000 population	·68
In rural areas of Hosur Taluq	1·91
In rural areas of Kollegal Taluq	3·36
In other rural areas	·10
In town circles of Bellary District	6·59
In rural areas of Bellary District	2·52

This gives a good idea of the manner in which the districts are affected. The main facts are—

(1) The rural areas of Kollegal Taluq, Bellary District, and Salem Taluq are the only parts of the Presidency in which rural plague has reached any degree of severity.

(2) In the rest of the rural areas of the affected districts plague has been extremely slight.

(3) In Bellary District the towns have suffered severely.

(4) In the other districts town plague has reached a fairly high level in a few large towns only, 8 of them accounting for most of the town plague. The other towns of these districts have been very slightly affected, an annual average of 395 deaths being scattered over 40 or 50 towns.

Thus, outside the Bellary District and the endemic areas of Hosur and Kollegal plague only reaches epidemic proportions which deserve consideration in a few big towns, eight of these towns having returned nearly as many deaths in a few epidemic as the whole of the rest of the infected districts with an area of over 60,000 square miles. Apart from these areas, (Bellary District and Hosur and Kollegal Taluqs) if it were not for the occurrence of occasional severe outbreaks in a few large towns, plague in the Presidency would have been of much less importance as a cause of mortality than such simple diseases as Diarrhœa and Bronchitis.

Having shown (*a*) the remarkable freedom of the lower-lying East Coast Districts of the Presidency from plague, (*b*) the presence of endemic centres in the small areas at over 3,000 feet elevation, (*c*) the occasional occurrence of plague, chiefly in epidemic form in large centres of population at moderate elevations, and (*d*) the frequent occurrence of severe epidemics in the elevated part adjoining Bombay Presidency it is necessary to show in what way the climatic conditions vary in these parts in order that it may be possible to consider whether the cooler climate of the higher levels has any direct effect on the distribution of plague.

The climatic conditions for the different parts of the Presidency will be best considered for the 5 main divisions which we have already made, and we have studied the temperature curves in these areas by means of 10-day normal curves made on the averages of a large number of years.

The striking characteristic of the first division, the East Coast Districts, is the absence of any well marked "cold weather" compared with what is found in other more northerly parts of India and in the more elevated parts of the South of India. Except in the most northerly parts of the East Coast area the so-called "cold weather" is of very short duration, the mean normal temperature for more than 9 months in the year being over 80 degrees. Even in the coldest months

the mean temperature does not reach much below 75 degrees in the whole of the area from Tinnevelly to Godaveri District. The "cold weather" does, however, become gradually more marked as one passes northwards and the most northerly districts of Vizagapatam and Ganjam show a well marked cold weather, the temperature in these districts reaching a lower level than in the Deccan area for part of the year. The contrast between the cold weather conditions in the southerly and low-lying part of the Madras Presidency and in say the Mysore Plateau or Bombay Deccan is very striking both as to duration and as to severity.

In the second area, the portions of the Central Districts at an elevation of a little over 1,000 feet, the mean daily temperature in the cold season is on the whole 2 or 3 degrees lower than in places in the low East Coast Districts at the same latitude. The incidence of plague in this area shows that it is not a very favourable place for the spread of plague, most of the mortality being due to the occurrence of a few epidemics in municipal towns.

The cold season in this second area is somewhat longer than in the first and it is particularly long in Coimbatore on account of its peculiar exposure to both monsoons.

In the highest districts of the Presidency which form part of the Mysore Plateau at an elevation of 3,000 feet, the climate is very mild all the year round. There is no very severe hot weather and the normal mean temperature varies from 68 to 82 degrees, the period during which it is over 80 being only a few weeks in the year. The temperature curve here very much resembles that of Belgaum, one of the severely affected districts of Bombay Presidency. No other part of the Madras Presidency, except the Nilgiris, presents such a long period of continuous moderately low temperature, or reaches such a low level.

The Bellary District and the adjoining portions of Anantapur and Kurnool which form part of the Deccan area have an annual variation of mean temperature of from 73° to 94°. The hot weather is very severe, but there is a moderate cold weather lasting for a little over 3 months. The temperature reaches a lower level than in most parts of the Presidency excepting the Nilgiris, the Mysore Plateau areas, and the northmost districts of the Circars. The difference in temperature from other less affected parts of the Presidency is not sufficiently great to be reasonably considered as an explanation for the special severity of the incidence of plague in the Bellary District. Mention has already been made of the importance of the proximity of this District to the affected parts of Bombay Presidency.

The fourth of our divisions, the West Coast districts, is characterised by the equability of the temperature throughout the year. The annual variation is usually within 10 dégrees—a few degrees above and a few degrees below 80°. Neither hot or cold weather is well marked and apparently not sufficiently so to produce any marked effect on plague when it is once established as in the case of Mangalore. The conditions do not appear to have been favourable to dissemination of plague or to its reaching any severe epidemic proportions.

On the Nilgiri Plateau at Wellington (Coonoor), at an elevation of 6,200 feet above sea level, the annual variation of temperature is from 56 to 67. In Ootacamund which lies 1,000 feet higher the temperature is even colder. In this climate we have prolonged existence of plague infection without much tendency to severe epidemics.

It is thus apparent that leaving aside for the present the Nilgiri Plateau with its temperate climate, plague is most continuously present and most persistent in the high Mysore Plateau, where there is the longest period of fairly low mean temperature. At lower levels, somewhat over 1,000 feet, where the temperature is higher and the cold weather shorter, plague is less constantly present and less persistent although when it once catches

hold it may occur in severe epidemic form for a limited season. At this elevation in different districts the severity of plague has generally been proportion to the proximity of the district to affected areas outside the Presidency.

Below 1,000 feet plague hardly ever occurs. The lower-lying East Coast Districts have no real cold weather, with the exception of the most northerly which are well removed from the centres of infection.

The West Coast Districts with no severe hot or cold weather have suffered little from plague except in Mangalore and the disease has neither been severe nor persistent in these districts except in that one town.

There is, then, a fair degree of correlation between climatic conditions and the incidence of plague in the Presidency with proximity to infected areas as a modifying factor.

RELATION OF FLEA PREVALENCE TO PLAGUE.

In other parts of India the Commission have found a remarkable correlation between the seasonal prevalence of fleas on rats and the seasonal prevalence of plague. This correlation has also been found to hold good in Madras Presidency in the places in which we have worked. We have also found differences in the flea-counts on rates in places presenting varying climates and varying plague incidence. Our main stations for observing the flea prevalence on rats were Madura, Coimbatore, Vaniambadi, Madras and Denkanikota. Madura and Madras represent the low-lying East Coast Districts, Vaniambadi the 1,000 feet elevation in the Central Districts where occasional outbreaks of plague occur, Denkanikota the 3,000 feet elevation on the Mysore Plateau in Hosur Taluq where plague is endemic. Coimbatore represents the peculiar conditions opposite the Palghat Gap.

In each of these places rats were caught daily and the average numbers of fleas per rat estimated. From these daily flea-counts fortnightly averages were calculated for a complete year.

The place showing the highest average number of fleas per rat throughout the year was Denkanikota. Here the fortnightly average varied from 3·1 to 14·9 and was only below 5 per rat for 3 out of the 26 periods. The flea-count maintained a fairly high level for a considerable part of the year. In Vaniambadi the fortnightly average varied from 1·9 to 10·4 and was above 5 for 14 of the fortnightly periods. For only two months of the year did the flea prevalence reach moderately high levels and this higher flea prevalence coincided with an outbreak of plague in the coolest months of the year.

In Coimbatore the flea prevalence varied from 1·4 to 10·4 and was above 5 for 15 out of the 26 periods. The fleas remained for a longer period at a moderately high level in Coimbatore than in Vaniambadi and reached a fair level at an earlier period. This earlier and longer period of high flea prevalence in Coimbatore corresponds with an earlier and longer season of plague prevalence.

In Madura the average number of fleas per rat was never so high as in the three cooler places. It varied from 3·5 to 6·8 and was only above 5 for 9 of the fortnightly periods.

The observations in Madras City were made in the previous year and the flea-count was somewhat lower than in Madura. It thus appears that the longest period of high flea prevalence occurs in the highest and the coolest of these places, in the area where plague is endemic. The lowest flea-count was made in the low-lying and hotter areas which have escaped plague. The places which occupy an intermediate position with regard to elevation and temperature also do so with regard to flea prevalence and are only occasionally plague-infected.

The areas not represented by these flea observing stations are Bellary District, the Nilgiris and the West Coast. Short observations in Bellary town in the cold weather have given flea-counts of 6·5 in October, 6·5 in November, and 7·5 in January, so the fleas per rat probably do not reach any great height here but maintain a moderate level for about 4 months. In the Nilgiris and on the West the flea-counts are usually low being as a rule under 5 per rat. In these areas we have special factors such as prolonged low temperature in the Nilgiris and high humidity on the West Coast which have important actions on the length of life of the rat flea and probably also on the spread of plague, but it is impossible to go into these matters in the scope of this paper.

These facts as to the distribution of plague in the Madras Presidency and its relation to geographical conditions, climate and flea prevalence, which have been put as shortly as possible here, show definite lines along which an explanation of the Madras Plague problem may be sought.

In the meantime this summary will form a basis for discussion by the Members of the Conference.

TABLE No. I.

Plague Deaths in Towns and Rural Areas in infected districts from 1901 *to* 1910.

Districts.	Population of town circles.	Population of rural areas.	Plague deaths in town circles.	Plague deaths in towns of over 10,000 inhabitants.	Plague death in rural areas.
Anantapur	111,500	810,332	689	266	2,264
Bellary	171,205	785,872	11,290	9,632	19,859
Coimbatore	160,254	1,835,933	3,881	3,718	8,088
Cuddapah	118,839	1,037,276	43	33	264
Kurnool	46,515	824,933	10	9	891
Malabar	218,007	2,567,300	912	912	135
Madura	339,015	1,493,134	357	357	496
Nilgiris	27,121	80,552	784	529	577
North Arcot	171,499	2,035,618	1,361	1,347	1,573
Salem	177,269	1,719,712	6,537	6,168	5,526
South Canara	52,149	1,082,240	2,613	2,611	299
Totals ...	1,593,373	14,272,902	28,477	25,582	39,972

Average annual death-rate per mille :—

Town circles ... ... 1·78 per mille.

Rural areas ... ... 0·28 „ „

TABLE No. II.

Average Annual Death-rate per Mille for Town Circles and for Rural Areas of the infected districts of Madras Presidency for the years 1901 *to* 1910.

Districts.	Town circles.	Rural areas.
Anantapur	·61 per mille	·27 per mille
Bellary	6·59 ,,	2·52 ,,
Coimbatore	2·42 ,,	·44 ,,
Cuddapah	·03 ,,	·02 ,,
Kurnool	·02 ,,	·10 ,,
Madura	·10 ,,	·03 ,,
Malabar	·41 ,,	·005 ,,
Nilgiris	2·89 ,,	·71 ,,
North Arcot	·79 ,,	·07 ,,
Salem	3·77 ,,	·32 ,,
South Canara	5·10 ,,	·02 ,,

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A Note on the Fleas infesting the Mus Rattus in Naini Tal and Mussoorie, by Captain H. Ross, M.B., I.M.S.

2 types of flea were found—

(*a*) Pulex Cheopis.

(*b*) Ceratophyllus Fasciatus.

During May and June :—Only 282 fleas were found on 291 rats, or less than one flea per rat.

182 of these fleas were microscopically examined and of these 107 were P. Cheopis and 75 C. Fasciatus, the proportion being 1·4 P. Cheopis to 1 C. Fasciatus.

July :—241 fleas were found on 198 rats examined=1·2 fleas per rat; thus during July the flea count per rat had slightly increased.

Of 240 fleas examined 196 were P. Cheopis and 44 C. Fasciatus, the proportion being 4·5 P. Cheopis to 1 C. Fasciatus, showing a considerable increase in P. Cheopis at the expense of C. Fasciatus.

Grain shop rats showed more fleas than houses in the proportion of 1·5 to 1; also a greater proportion of P. Cheopis, *viz.*, 6·8 to 1, as against 4·6 to 1.

August :—525 fleas were found on 169 rats=3·1 fleas per rat—a large increase.

This increase was chiefly in the number of P. Cheopis, which were found to be in the proportion of 12 to 1 C. Fasciatus. Grain shop rats however showed a smaller relative increase than house rats—only 2·7 fleas per rat as against 4 fleas per rat on the house rats.

House rats showed P. Cheopis to have increased out of all proportion to C. Fasciatus=21·6 to 1.

Grain shop rats showed 8·3 P. Cheopis to 1 C. Fasciatus.

September :—558 fleas were found on 123 rats=4·5 fleas per rat. The proportion of P. Cheopis to C. Fasciatus being 7·4 to 1, thus C. Fasciatus showed a proportional increase.

Grain shop rats showed a considerable increase in fleas=5·3 per rat.

House rats showing 3·7 fleas per rat.

Grain shop rats showed 17·4 P. Cheopis to 1 C. Fasciatus while the house rats showed only 3·7 P. Cheopis to 1 C. Fasciatus; thus there were considerably more C. Fasciatus on house rats than on grain shop rats.

October :—910 fleas were found on 284 rats=3·2 fleas per rat, the proportion of P. Cheopis to C. Fasciatus being 6·6 to 1.

Grain shop rats showed 3·2 fleas per rat, other shops 2·8; P. Cheopis was 12 times as numerous as C. Fasciatus.

In house rats the proportion was 4 P. Cheopis to 1 C. Fasciatus.

The rats caught in fruit and vegetable shops showed in all cases a greater proportion of C. Fasciatus.

Last Week in October :—Fleas per rat had gone down considerably, being only 1·6 fleas per rat.

The proportion of P. Cheopis to C. Fasciatus being now only 4 to 1.

Month.	Fleas per rat.	Proportion of Pulex Cheopis to Ceratophyllus Fasciatus.
May and June	·9	1·4 to 1
July	1·2	4·5 to 1
August	3·1	12 to 1
September	4·5	7·4 to 1
October	3·2	6·6 to 1
Last Week Ootober	1·6	4 to 1

In Mussoorie all the fleas found on the Mus Rattus were Ceratophyllus Fasciatus and the flea count per rat rose to only 2·5 in the rains.

In Naini Tal the flea counts being now obtained show that the increase which occurs in the rainy season August-September and early October is a very temporary one, falling again quickly considerably below the figure required to carry on a severe epizootic.

It will be necessary to carry on the experiment until next May, but during the period (August to October 15th) in which the flea count is probably sufficient to carry on a mild epidemic. Naini Tal is not at all liable to become infected owing to the absence of plague from all districts surrounded. It is interesting to note that 3 cases of plague were introduced into the most crowded bazaar in Mussoorie in September-October, but the disease failed to spread, the rat flea count at the time being about 2 per rat.

Everything appears to point to the fact that the rat flea infestation in these stations is below that necessary to carry on a severe epizootic, thus precluding the possibility of a severe epidemic of plague.

ALL-INDIA SANITARY CONFERENCE—MADRAS—1912.

THE PREVENTION OF PLAGUE.

By

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The commonly accepted measures, which are generally adopted by medical officers for the prevention of palgue, are disinfection, rat killing, and inoculation. In a preceding paragraph I have discussed the value of disinfection by bactericidal disinfectants and have pointed out the futility of using them. I now propose to discuss the value of disinfection by pulicidal or combined pulicidal and bactericidal disinfectants, rat killing and inoculation, from the larger point of view of the epidemiologist and sanitarian applying his measures to provinces rather than from the standpoint of the medical officer who is applying his measures to single villages or towns.

The use of pulicidal or combined disinfectants merely serves the same purpose as evacuation: it gets rid of the fleas from the premises and makes them habitable again, and it does so more quickly than can be effected by evacuation. The circumstance that the fleas are killed by the disinfection possesses little, if any, importance and offers no advantage, for that reason, over the slower process of evacuation, because fleas are short lived and multiply with enormous rapidity and the mere existence of large numbers of fleas is comparatively unimportant, provided that they are not in human habitation, and are not infected.

It is however a matter of importance to remember that the disinfection disposes of the fleas for the time being only and does not cut off the source of supply of more fleas, and the danger of relying on disinfection depends on this,—that it gets rid of the fleas at once and apparently allows of immediate reoccupation of the premises: but nothing could be more dangerous: as soon as the house is again inhabited the rats will return and the conditions favouring recurrence of human plague again prevail almost immediately. The slower method of evacuation is therefore more reliable and more scientifically certain, and disinfection should not be greatly used nor injudiciously lauded, because it is not really reliable as a means of protection, and its failure recoils upon the authority advising it and causes lack of confidence in other sanitary measures.

With regard to rat killing, I admit its utility in small areas such as one's own compound or possibly in a small village, but I argue that its general application is impossible and futile, and therefore its utility is so limited as to render it an insufficient and unsatisfactory preventive measure.

I have myself superintended rat killing operations of an experimental nature on a large scale where over one million rats were killed in three years in a small town and its neighbouring villages. The immediate effect was good but within a few months from the cessation of operations, plague was again active and prevalent—and further, the cost was enormous, working out at about Re. 1 per head of protected population per annum without counting any charges for supervision.

The explanation seems to me to be the same as that which accounts for the fact that there are still plenty of fish in the sea in spite of all the efforts of man and steam trawlers, which latter were at one time accused of being in process of killing off the supply for good and all. It is merely to admit that all man's efforts are futile, when he ranges himself against the vast reproductive powers of Nature, which are so adjusted, or adjustable as to maintain a constant stock in proportion to the available food supply.

With regard to inoculation, I not only admit its efficacy in the individual, but consider that it is an important preventive factor in communities; but I would lay stress upon the points in its use which are commonly slurred over or ignored.

In the first place the material is a "vaccine" and should be used as such, and its dosage should be more scientifically standardised and measured and it should be administered in a series of increasing inoculations in order to obtain the maximum benefit. The present dosage is most unsatisfactory and frequently produces profound reactions which advert against its use, and probably produce deferred and diminished immunity in the cases where they occur.

In the second place the immunity produced is transient and short, and is even shorter and less potent than it would be if the dosage were better standardised and the vaccine inoculated in a series of doses.

The period of efficacy is unknown but may be demonstrated as a curve, rising sharply for the first few days and more slowly up to the 8th or 10th day after which it gradually drops over a period of unknown duration, but probably of not more than 3 or 4 months, as far as practical benefit is concerned, and under the existing system of inoculation.

The degree of immunity is also unknown but is admittedly only partial.

The benefit is only an individual one and does not, like the acquired immunity against small-pox, in any way protect any one else, and it is largely because of this fact, and the transient nature of the immunity, that inoculation is not a preventive measure of great value.

In this connection it is a matter of importance to remember that the disease is not generally a contagious one, and that the infection is indirectly obtained from the rat, and that therefore the protection of the individual human being does not help to protect the race, because it does not reduce the number of possible sources of infection.

When we consider the differences in the nature and means of infection, and in epidemiology, between plague and other epidemic diseases, we must recognise the primary importance of these points and therefore we should attack the problem of prevention on other lines, not on the same lines.

We have a chain of associated factors in which we only require to break one link to prevent the occurrence of epidemic plague, and it seems to me that the lesson we may learn from the occurrence of epidemic rat plague in England in several places, and to a large extent without the outbreak of human plague in epidemic form, is the lesson from which we are likely to evolve the real means of permanent prevention.

In the chain of infection we have the rat, the flea and man. It seems to me that the explanation of the failure of plague in England to become epidemic is due to the disassociation of man and the rat, and the consequent difficulty of access of the rat-flea to man. This separation is largely fortuitous, and is due to the differences in habit and habitation of the two animals, rat and man. The flea may be left out of the argument because, although it is admittedly present, it cannot cross the gulf between the two hosts.

The differences in habit and habitation referred to are probably accidental, and due to differences in climate and to the higher standard of comfort called forth by the increase in wealth, and the progress in education, which have been made in the past century or two.

But the important point is that these conditions of difference can be imitated, and it is by their imitation that we are most likely to prevent plague. Let us therefore examine these differences and see what are the general principles underlying them.

It seems to me that the most important differences in the habitations are that houses in England are of masonry, and are largely rat proof, and that the drains are not open high ways for the passage of rats, but are closed and trapped, so that the rats and men are kept separate by a water seal and the circumstance that the houses are *pucca*.

In the matter of habit there are also differences, in which the chief point seems to be that the rat is not so domestic an animal in England as he is in India—probably because of the difference in the construction of the houses and drains, which has driven him into a different mode of life. At any rate the fact remains that in India the rat lives in the roofs and walls of houses and feeds on the waste food of the people which is left lying about or is thrown away, whilst in England the rat lives outside houses, either in sewers, or in ware-houses, or stack-yards and barns, and lives as a beast of prey upon whatever he can get, but he does not habitually frequent houses nor does he depend on the human host for refuse food.

THE ABSENCE OF A "NEGATIVE PHASE" AFTER INOCULATION WITH PLAGUE PROPHYLACTIC.

BY

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INDIAN MEDICAL SERVICE.

The question of the existence of a negative phase after inoculation with plague vaccine has again risen since the discussions of the delegates to the International Plague Conference at Mukden in April 1911. By a 'negative phase' I mean a period of definite hyper-susceptibility to plague infection and not merely a latent period during which no immunity is being produced.

During the discussions it was obvious that the opinions of the delegates were divided on the subject. That it is one of great importance every inoculator will agree. Can the vaccine be safely administered to persons living in plague-infected houses, and again, an aspect of the problem that especially puzzled the workers in Harbin according to Dr. Wu, a Chinese delegate, what length of time should elapse between inoculation of the plague staff and the date they are allowed to start work among plague cases? This, of course, is of special importance in an epidemic of Pneumonic plague where transmission of the infection to attendants is so common.

On reading the official report of this Conference, one is struck by the lack of any data from which it might be possible to deduce observations. Dr. Paul Haffkine, for example, Director of the Russian Plague Hospital at Harbin, talks of the "usual period of the negative phase which is 12 days." *But when asked by Dr. Richard Strong, the Professor of Tropical Medicine at Manilla, to give his evidence, he replies, "The human body is weakened by inoculation. I only found 4 cases in which plague was contracted 12 days after inoculation. All the others who got infected were infected in the first days after inoculation. Therefore it seems probable that there is a negative phase."

He seems to have forgotten that those who, he says, were infected in the first days after inoculation, may have been incubating the germs before they were inoculated at all. He frankly stated when pressed that his opinion was an "hypothesis."

The general opinion of the Conference was that the subject required further evidence and I propose to give such evidence now.

Before I do so, however, let me detail the previous literature and experiments on the subject.

In a paper entitled "A discourse on Preventive Inoculation" delivered* at the Royal Society, London, on June 8th, 1899, Haffkine of the Plague Laboratory, Bombay, stated that his experience in anti-cholera inoculation entitled him to give a reassuring answer to this question.

From the Undhera statistics Haffkine deduced:—

> "Inoculation has again acted, so to say, immediately; or as we have adopted to generally formulate the result, has acted within the time necessary for the subsidence of the general reactionary symptoms produced by the inoculation."

* On page 124.

Bannerman in the "Proceedings of the Royal Society of Edinburgh," 1901, Vol. 24, Part 2, to which paper we are indebted for reference to much of the literature on this subject, also states that:—

> "Protection begins to be effective after the lapse of 24 hours and goes on steadily increasing for some considerable number of days thereafter."

On the other hand, we have the views of the Indian Plague Commission Report of 1898-99, Vol. 5, page 262, that inoculation does not appear to confer any such degree of protection within the first few days after the inoculation has been performed.

We can find references to only two animal experiments on the subject:—

First.—Calmette's experiments recited in brief in the British Medical Journal of October 27, 1900:—

> "With M. Salimbeni he had shown during the recent epidemic in Oporto, that animals during the period of immunisation with heated cultures were extremely sensitive to very small doses of the virus, doses which were rarely mortal to non-vaccinated animals. It followed that a person in the period of incubation for a slight attack of plague would find the disease considerably aggravated if he submitted during this period to a preventive inoculation of Haffkine's vaccine. The case would almost certainly be fatal."

The cultures used by Calmette and Salimbeni were killed at a temperature of 70° C. and therefore were of little, if any, immunising value, as it is well-known that such a temperature to a large extent destroys the plague toxin. The dose given to mice was one half cubic. centimeter and 1 to 2 cubic centimeters for guineapigs. These writers stated that with such a dose "immunity only establishes itself at the end of 8 to 10 days and scarcely lasts more than 2 weeks."*

According to Professor Zabolotny, Professor of Bacteriology, Medical Institute, St. Petersburg, "Calmette's investigations were only done with a few animals." Report of International Plague Conference, Mukden, 1911. I cannot find out how many animals were used.

Second.—Some experiments were reported in the Bombay Bacteriological Laboratory's Annual Report for the year ending 31st March 1905. There, the uncertainty of the knowledge at that time about this question of "negative phase" was expressly implied. It is pointed out that prior to certain experiments there detailed, carried out by Captain Liston, I. M. S., the dose of prophylactic for persons living in infected households was half that for others not so in contact with plague.

These experiments, however, although they were held to be sufficient evidence to justify the administration thereafter of the same full dose to those living in infected houses as to those not in contact with plague conditions are not conclusive, (1) because of the small number of guineapigs experimented upon, and (2) because the average number of days the animals lived after inoculation with plague is taken as the index of the immunity produced by the prophylactic. Of this assumption there is no proof, although it is probably correct that prolongation of the incubation period of the disease indicates an increase in the acquired immunity to the disease.

I will now detail two fresh experiments carried out by us in the months of March and April 1911.

Both the experiments were made on rats (*mus rattus*) caught in Madras. These rats the Plague Research Commission have shown are highly susceptible to plague differing in this respect from the rats now procurable in Bombay.

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* Annales de l'Institut Pasteur, Tome XIII, 1899.

Experiment I.—The total number of rats experimented with, namely, 280, was divided up into 8 groups of 35. The plague prophylactic vaccine employed throughout the experiment had the following history. The source of the bacilli was from human plague passed through a guineapig. It was brewed at room temperature for two months. It was kept in the cold room in darkness from 24th February 1911, the date of manufacture.

One "A" group received on 30th March 1911, 14 days before it was inoculated with plague	0·25 C. C. of the vaccine.
One "B" group received on 3rd April 1911, 10 days before it was inoculated with plague	do.
One "C" group received on 4th April 1911, 9 days before it was inoculated with plague	do.
One "D" group received on 6th April 1911, 7 days before it was inoculated with plague	do.
One "E" group received on 8th April 1911, 5 days before it was inoculated with plague	do.
One "F" group received on 10th April 1911, 3 days before it was inoculated with plague	do.
One "G" group received on 12th April 1911, 1 day before it was inoculated with plague	do.
One "H" group was kept as a control receiving no treatment.	

Certain of these rats in the various groups died from handling or effect of the toxin, leaving the following numbers in the various groups alive on the 13th April 1911:—

A	B	C	D	E	F	G	H
32,	33,	34,	29,	33,	33,	35,	35.

On the 13th April all these groups received simultaneously the same dose of plague,—namely, an emulsion of spleen from a rat which died of plague, the spleen smears showing large number of plague bacilli. Each rat in these groups received ·0019 of a milligramme of the plague spleen.

Every rat which died was sectioned and smears from the various organs examined for plague bacilli, and unless these were found the death was not ascribed to plague.

At the close of the experiment the following number of rats remained alive in each group :—

A	B	C	D	E	F	G	H
14,	12,	12,	11,	15,	20,	13,	4.

The following table gives the incidence of deaths in the various groups day by day.

Table I.

Date on which death occurred.	A Group.*		B Group.		C Group.		D Group.		E Group.		F Group.		G Group.		H Group.	
	Not plague.	Plague.	Not plague.	Plague.	Not plague.	Plague.	Not plague.	Plague.	Not plague.	Plague.	Not plague.	Plague.	Not plague	Plague.	Not plague.	Plague.
5th April 1911	.....	.....	1*	.....	1*	.....	.....	.....	2*	.....	.....	.....	.....	.....	.....	1
16th „ „	.....	.....	.....	.....	.....	1	.....	1	.....	3	1*	.....	.....	.....	.....	4
17th „ „	.....	7	.....	4	.....	4	.....	3	.....	5	.....	3	.....	3	.....	11
18th „ „	.....	3	.....	4	.....	6	.....	3	.....	.....	.....	1	.....	8	.....	6
19th „ „	.....	2	.....	5	.....	4	.....	5	.....	.....	.....	2	.....	3	.....	6
20th „ „	.....	1	.....	1	.....	4	.....	3	.....	.....	.....	.....	.....	2	.....	1
21st „ „	.....	1	.....	.....	.....	.....	.....	.....	.....	3	.....	2	.....	4	.....	.....
22nd „ „	.....	.....	.....	3	.....	.....	.....	1	.....	1	.....	1	.....	2	.....	.....
23rd „ „	.....	2	.....	3	.....	2	.....	2	.....	1	.....	3	.....	.....	.....	.....
24th „ „	.....	2	.....	.....	.....	.....	.....	.....	.....	3	.....	.....	.....	.....	.....	2
Totals ...	18		20		21		18		16		12		22		31	
	Out of 32.		Out of 32. 1* not counted in total numbers.		Out of 33. 1* not counted in total numbers.		Out of 29.		Out of 31. 2* were not counted in total numbers.		Out of 32. 1* not counted in total numbers.		Out of 35.		Out of 35.	

It will be seen that even in the group "G," which received the vaccine one day before inoculation with plague, the number of deaths is less than in the control group "H," which received no prophylactic treatment.

On the whole the best result is in group "F," which was inoculated with plague three days after receiving the dose of vaccine.

Experiment II.—The total number of rats experimented with was 280, divided up into 7 groups of 40 rats each. The same vaccine was employed as in Experiment I. One group was kept as a control—receiving no preliminary prophylactic dose. The remaining 6 groups were treated as follows :—

One "A" group received on 25th April 1911, 3 days before inoculation with plague	0·25 c. c. of vaccine.
One "B" group received on 26th April 1911 (5 p.m.), 46 hours before inoculation with plague ...	do.
One "C" group received on 27th April 1911 (5 p.m.), 22 hours before inoculation with plague ...	do.
One "D" group received on 28th April 1911(6-30 a.m.), 8½ hours before inoculation with plague ...	do.
One "E" group received on 28th April 1911 (10 a.m.), 5 hours before inoculation with plague ...	do.
One "F" group received on 28th April 1911(1-30 p.m.), 1½ hours before inoculation with plague ...	do.
One "G" group, control rats, received no prophylactic.	

Certain of the rats in the various groups died from handling or the effect of the toxin, leaving the following numbers alive at the time of inoculation with plague at 3 p. m. on the 28th April 1911 :—

A	B	C	D	E	F	G
39,	40,	39,	40,	40,	40,	40.

At 3 p. m. on the 28th April, all the groups were inoculated with doses of an emulsion of a plague rat spleen, each dose containing ·0009 of a milligramme of spleen-tissue.

At the close of the experiment, the following number of rats remained in each group :—

A	B	C	D	E	F	G
11,	20,	14,	14,	9,	7,	6.

The following table shows the incidence of deaths in the various groups day by day (Table II attached).

Conclusion.

These two experiments show conclusively (1) that there is no "negative phase" or period of increased susceptibility of rats to plague after the administration of anti-plague vaccine even within one and half hours after the operation. From recent work done by the Plague Commission in England showing that vaccines which immunise guineapigs are not efficacious in an equal degree in the case of rats and *vice versa*; and in view of our ignorance of the methods by which immunisation is effected, it would be unwise to confidently apply these results of experiments among rats to immunity work among human beings. Taken, however, in conjunction with the deductions drawn from a huge number of statistics of inoculation work in India, it is probably safe to state that inoculation is not only harmless, but in fact beneficial to persons living in actual contact with plague conditions. (2) That the production of immunity among rats commences immediately and increases in amount till the 2nd or 3rd day after anti-plague vaccination. It then declines somewhat in amount and remains more or less steady from the 4th day onwards to the 14th—as far as our experiments have been conducted.

W. D. H. STEVENSON, M.A., M.B.,
CAPTAIN, I.M.S.

Assisted by

KHAN BAHADUR SENIOR ASSISTANT SURGEON
A. J. KAPADIA.

Table II.

Date on which death occurred.	A Group.		B Group.		C Group.		D Group.		E Group.		F Group.		G Group.	
	Not plague.	Plague.	Not plague.	Plague.	Not plague.	Plague.	Not plague.	Plague.	Not plague.	Plague.	Not plague.	Plague.	Not plague.	Plague.
29th April 1911	.....	.....	1*	.....	2*	.....	.....	.....	1*	.....	.....	.....	.....	.....
30th „ „	.....	.....	.....	.....	.....	.....	.....	1	.....	.....	.....	.....	.....	.....
1st May „	.....	1	.....	2	.....	1	.....	2	.....	.....	.....	2	.....	5
2nd „ „	.....	4	.....	6	.....	7	.....	4	.....	7	.....	8	.....	7
3rd „ „	.....	9	.....	4	.....	5	.....	10	.....	9	.....	10	.....	6
4th „ „	.....	4	.....	1	.....	3	.....	5	.....	5	.....	4	.....	5
5th „ „	.....	3	.....	2	.....	1	.....	3	.....	5	.....	2	.....	6
6th „ „	.....	4	.....	2	.....	1	.....	.....	.....	1	.....	2	.....	3
7th „ „	.....	1	.....	2	.....	4	.....	1	.....	3	.....	3	.....	.....
8th „ „	.....	2	.....	.....	.....	1	.....	.....	.....	.....	.....	2	.....	2
Totals ...		28		19		23		26		30		33		34
		Out of 39.		Out of 39. 1* not counted.		Out of 37. 2* not counted.		Out of 40.		Out of 39. 1* not counted.		Out of 40.		Out of 40.

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PLAGUE PREVENTIVE MEASURES.

BY

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In seeking for a subject for discussion at this Conference I have chosen the somewhat general theme "Plague Preventive Measures." I have been guided in my selection by the thought that we are here to confer with one another, to suggest means for combating disease, and to record our experience of the success or failure of measures designed to prevent human suffering. We are here to learn from one another what difficulties have been encountered and it may be how these difficulties have been overcome in some cases.

While it is true that I have had little practical experience in the prevention of the spread of plague I have been fortunate in having had unrivalled opportunities, in association with my colleagues of the Plague Research Commission, for studying this disease in many parts of India. I trust this training will make up for my lack of administrative experience when speaking to you on plague preventive measures.

In a paper which I read before the Bombay Medical Congress in 1909 on the Prophylaxis of plague I stated that preventive measures against this disease must be based on a knowledge of its ætiology if these measures were to be successful. I described plague as essentially a rat disease and in the respect that man participates in the disease more or less as he comes in contact with rats I likened plague to such diseases as hydrophobia where dogs are largely responsible for human infection, or glanders where horses generally convey the disease to man, or anthrax in which case horned cattle spread the disease. Measures to prevent the spread of these diseases to man are designed to prevent the spread of the disease among the animals affected in each case. I refer to such measures as the muzzling orders for dogs to prevent the spread of hydrophobia, or the destruction of glandered horses or anthrax infected herds. In a similar manner in the case of plague, measures against this disease must in the first instance be designed to prevent the spread of the disease among rats. In the cases of such diseases as hydrophobia, glanders, or anthrax wholesale destruction of the animals responsible for the spread of these diseases is often resorted to and this is effected by the aid of the law; competent authorities are empowered to seize and destroy without compensation valuable animals; the individual interests of the owners are seldom considered.

Plague is spread to man by a worthless, often destructive animal, yet, in this case the sanitary authorities have no legal power to enforce the destruction of rats and much greater consideration is shown to the interests and even the prejudices of individuals than when valuable animals are concerned as I have shown above. Persons who harbour rats on their premises are not compelled to get rid of them or take the precaution, if they have scruples in the matter of their destruction, to prevent their migration to other premises. The absence of any law in regard to the harbouring of rats I venture to think is one of the weakest points in our present plague policy. It is often impossible to cause people to destroy rats which are not only a source of loss and danger to themselves but are frequently a danger to others. Be it noted that the danger of rats spreading plague to man is much greater than the danger of dogs carrying hydrophobia, or horses glanders, or cattle anthrax. Laws exist to prevent the lesser dangers but none exist for the greater. The Health Officer of Bombay City for example although he knows that certain godowns swarm with rats which are deliberately fed and watered by the owners of these godowns is yet unable either to compel the owners of the godowns to destroy the rats or make their godowns rat-proof nor can he on his part take action himself to destroy the rats. Is it not time that the hands of the sanitary authorities were strengthened by law to enable them to compel persons to destroy any rats they may harbour on their premises or at least cause them to make their property secure against rats?

The conditions which encourage the presence of rats encourage the spread of plague : chief among these conditions are an ample food supply for the rats, shelter to breed in, associated with the absence of enemies. The abatement of the conditions which favour the presence of rats must form the basis of our plague preventive policy.

An abundant food supply for rats can easily be found in every Indian town and village chiefly because the system of scavenging, where it exists at all in India, is defective. We devote much attention to the perfecting of systems of water-supply and drainage and we spend vast sums on their improvement but we pay little heed to scavenging ; we are generally content to leave that matter in the hands of ignorant sweepers. I am glad to see from the list of papers to be read at this Conference that we are to learn something about this matter from the Health Officer of this City, Dr. MacDonald, the subject is fortunately not to be altogether neglected as is usually the case. I do not wish to blame the sanitary officers for neglecting to pay more attention to systems of scavenging for I recognise that their task is a difficult and disagreeable one. The people of this country are so callous in regard to the accumulation of dirt and so indifferent to disorder that they do not realise the advantages of cleanliness and tidiness. They are in the habit of throwing waste food and other material anywhere so long as it does not remain in their dwellings. The most sanitary buildings are often defiled by the dirt which is thrown out of the windows. As I write this I recall to mind a visit I paid to a sanitary chawl erected by the Improvement Trust of Bombay which had been defiled in this way, in consequence it had been invaded by rats and infected with the plague.

In some cases matters are not improved by the fact that cattle, goats and hens are accommodated in dwelling houses. There may be some shadow of excuse for the presence of these animals in the houses of villagers where police arrangements are still imperfect but in towns, where this is still a common practice, the housing of these animals in human dwellings should under no circumstances be allowed. I am pleased to know that Dr. Turner, the Health Officer of Bombay, has pressed upon the Municipal Corporation the advisability of amending the Municipal Act so that no person shall "keep in any building any portion whereof is used or intended to be used for human habitation any horse, cow, buffalo, bullock or goat." I think it would have been well had poultry been included in this list.

Another source of food supply for rats in dwelling houses in towns are the grocers' shops in which grain and other food is stored in open baskets or other receptacles. I understand that Dr. Turner strongly urged upon the Municipal Corporation that the proximity of such shops to human habitations encouraged the presence of rats which are a danger to the inhabitants of the dwellings in which these shops are situated. Unfortunately it does not seem possible in the present circumstances to attempt to prevent the establishment of grocers' shops on the ground floors of inhabited houses, but the prohibition to the storing of grain in larger quantities than that required for retail sale in such situations has met with the approval of the Municipal Committee appointed to consider amendments to the Municipal Act. I hope it may yet be possible even in the case of retail grain dealers, to insist that, when their shops are closed, their wares shall be stored in rat-proof receptacles not in open baskets as is generally the case in Bombay city. I will be interested to learn what is being done on these lines in other towns than Bombay.

The importance of the grain trade in the spread of plague has been sufficiently dwelt upon by Captain White in his paper ; I need, therefore, say no more on this subject, but urge upon the Members of this Conference the necessity of attending to grain shops and grain godowns to see that they are made rat-proof by their owners, if necessary under legal compulsion. The arrangements in connection with the weekly markets which are so commonly held in small towns and large villages should not be overlooked, for these markets afford a

very favourable opportunity for the diffusion of plague. Recently Messrs. Mackenzie Brothers of Bombay prepared plans for me of a rat-proof market place which, I hope, will be built in Belgaum and perhaps also in Ballia. A central paved area is surrounded on four sides by ferro-concrete godowns, the doors of these godowns are supported on a steel framework with panels of thin steel and they are hinged, so that when opened they form a chabutra or shade to the small verandah in front of the godown. Everything has been arranged to make these godowns as attractive as possible to the merchants who will occupy them, so that they may be persuaded, if not compelled under the Epidemic Diseases Act, to evacuate their dilapidated rat-ridden premises in the midst of the town and occupy the pucca market godowns on its outskirts. The old grain godowns, I hope, will be acquired by the Municipality and pulled down and the land thus cleared used either as sites for depôts or stores or preferably planted with trees and left open for play-grounds for the children who at present have to be content with accommodation on the streets.

In addition to food rats require shelter to breed in, they are nocturnal in their habits and therefore prefer dark places to live in. Well lit and ventilated rooms are not selected by rats, but such rooms make the best quarters for men. Attention to the construction of human dwellings is therefore a very important plague preventive measure. So far as I am aware little has been accomplished in this direction. In certain towns it is true Improvement Trusts have been established and these have done admirable work. No one, for example, who has lived in Bombay during the past fifteen years can have failed to observe the splendid improvements effected by the Bombay Improvement Trust. The work of the Trust has been frequently criticised, but the difficulties which they have had to face, often without experience to guide them, have been enormous. The officers of the Trust are fully aware of their shortcomings, but unfortunately their hands are tied. The Hon'ble Mr. Orr, Chairman of the Bombay Improvement Trust, in an admirable lecture on "Light and Air in dwellings in Bombay" delivered before the Bombay Sanitary Association has fully explained some of the difficulties encountered by the Trust. I strongly commend the perusal of this lecture to Members of this Conference and I cannot do better than make a few extracts from it here. In the first place, Mr. Orr recognises that although the Trust has done much "towards wiping insanitary rooms out of existence by wholesale demolition of houses in insanitary areas," "such campaigns," he says, "have been extremely expensive and have often, when demolition has been too rapid, resulted in the increase of overcrowding in the immediate neighbourhood." Mr. Orr is of opinion that the improvements made by the Trust "in a few small sections of the city are as a drop in the ocean to the rapid advance in insanitary conditions which is going on all over the city by virtue of the want of some check on extensions of buildings outwards and upwards and the consequent steady deterioration of the already defective lighting and ventilation of the quarters inhabited by the poorer classes of the population." "Laudable efforts," he adds, "are being made to relieve sufferers from tuberculosis by providing hospitals and sanitaria for them, but that strikes me as attacking the problem at the wrong end. Far better go to the root of the matter and make hospitals and sanitaria unnecessary by first protecting people from living in dark foul dens, which serve as foci for the propagation of tuberculosis and other diseases, and then wiping out some of these dens in order to convert the others into healthy residences by the admission of the two great healing powers nature supplies gratis—light and air." I quite agree with Mr. Orr's opinions, I wish only to emphasise them here and to say that they are as applicable to plague as to tuberculosis. Mr. Orr points out that although the Municipal Corporation took ten years to discuss and frame the present Building Bye-laws they are still very defective in that they do not insure the proper lighting of back rooms and side rooms, the letter of the law is generally though not always strictly adhered to, but the spirit of the law is evaded. But even if these Building Bye-laws are now amended their effect will only be obvious in the remote future and what is necessary now is some power to rectify, not only avoid, the mistakes of the past. He proposes

as a first step towards mending matters legislation prohibiting the use for human habitation of rooms that fail to satisfy the 63½° standard of lighting and ventilation, such prohibition to apply generally all over the city after five years, but to be meanwhile applied to the worst rooms in specially insanitary areas, temporary accommodation being offered to the tenants till the landlords have rendered their rooms fit for human habitation. These are very excellent suggestions which have the great merit as I have tried to indicate that they not only avoid errors in the future but also rectify the errors of the past. They have the additional recommendation that they have been carried out with success over a limited area which is in the hands of the Trust. I hope that powers may be granted to the proper authorities to carry out Mr. Orr's scheme for securing more light and air in the dwellings of the poor. His suggestions are eminently practical and based on extensive experience.

In the third place the absence of the enemies of rats encourages their multiplication. The most important enemy of the rat in civilised countries is man, but here people are ignorant of the diseases caused by rats and of the loss that they occasion. It is very necessary therefore to enlighten the people and stimulate a hatred and loathing for these animals such as exists in Europe. This cannot be done unless an attempt is made to enlighten the people in a much more systematic manner than at present. It is no person's particular business to impart popular instruction in connection with plague or other matters pertaining to public health. There is a want of system in getting at the people, they are generally told about diseases when they are panic-stricken and afflicted by them. In a word our efforts to teach the people how disease is spread and how it may be prevented are not sustained. The knowledge is imparted by an untrained agency at an inopportune moment. How are these defects to be remedied? Here, of course, we are confronted at once with the question of expense. A complete medical and sanitary organisation which would be in close touch with the people, always diffusing information regarding the prevention of disease and actively employed in the preservation of the public health, would be ideal. Such an organisation would be responsible for the registration of births and deaths, the carrying out of vaccination and the arrangements connected with epidemics, famine, etc. This staff, for example, would be responsible for inoculation, evacuation and rat destruction in connection with plague epidemics. It would be a permanent organisation and the staff would as far as possible be recruited and maintained in the areas in which they worked, so that they would be known to the people. A reserve staff would be maintained which could be drafted to areas of greatest pressure. The supervision of the staff would be in the hands of the district officers, medical and civil. It is probable that at present such a scheme would be financially impracticable, but even now with better organisation something can be done at less cost to improve the present methods of communicating information on hygiene to the masses.

In the first place, some department should be held responsible for the diffusion of knowledge regarding the spread and prevention of disease. It seems reasonable that a department actively engaged in research of this nature should be the department selected for the purpose. In other words, a bureau for the collection and diffusion of knowledge in regard to preventive medicine should be associated with each Research Laboratory.

In the second place a suitable staff should be provided for the purposes of the bureau. Persons engaged in research are not always the most fitted to communicate their findings to the public. Success in imparting information depends largely on the manner of its presentation. Persons should be selected for this purpose who have a facile pen and a gift of making the subject on which they write simple and interesting. If possible these accomplishments should be associated with the power to organise.

In the third place the department responsible should be furnished with funds for the purpose of popularising a knowledge of Hygiene. The preparation of books and pamphlets well illustrated would constitute a part of the duties of the bureau.

In the fourth place an attempt should be made to utilise each and all the following existing organisations for the diffusion of knowledge so that those who can read may communicate the information to the illiterate :—

(1) The Revenue Department.
(2) The Medical and Sanitary Departments.
(3) The Police Department.
(4) The Educational Department.
(5) The Postal Department.
(6) The District and Taluq Boards.
(7) Village Sanitation Committees (Bombay Act of 1889).
(8) The Railways.
(9) Agricultural, Co-operative and other Societies.
(10) The Press.
(11) Private Medical Practitioners, Hakims and Vaids.
(12) Priests, Mullahs and Missionaries.
(13) Markets and Fairs.

In the fifth place centres for teaching by demonstrations and lectures should be established. We have arranged such demonstrations at the Parel Laboratory and I derive some pleasure in knowing that those demonstrations have had some effect in bringing about the amendments to the Bombay Municipal Act referred to above in connection with the housing of cattle in dwellings and the regulations regarding grocers' shops.

I have purposely enlarged on what I have called in another place the permanent measures for the prevention of plague ; these measures have the recommendation that while they are applicable to plague they are also useful for the prevention of other diseases. I have left myself little time to speak of temporary plague preventive measures such as rat destruction, inoculation and evacuation. These measures have been so frequently discussed that I need say little about them here. As regards inoculation, however, I have only to remark that its popularity is increasing, and it is becoming more and more recognised that this is the cheapest, most efficient and best tempoeary method of dealing with plague in the midst of an epidemic. It is re-assuring to know from Captain Stevenson's work that there is no danger in undergoing inoculation in the midst of infection.

One point remains to be touched upon and it is this that rats are harmless so far as plague is concerned if they have not infection amoung them so that where rats are numerous and action cannot immediately be taken to exterminate them every precaution should be adopted to insure that infection is not brought to them. The prevention of the importation of infection is a very important plague preventive measure. I wish therefore to draw attention to Captain Kunhardt's paper in which he shows that apart from the large towns with over one hundred thousand inhabitants, plague lingers in villagesduring the quiescent plague period or off-plague season. Such villages generally become infected late in the active or epidemic plague season and are the centres from which the disease is spread in the following epidemic season. The larger the number of these late infected villages in a district which carry on the infection during the quiescent plague period, the severer is the disease in that district during the following active or epidemic season. When no villages carry on disease during the quiescent period a disriot is likely to escape infection in the following epidemic season. This goes to prove

that infection is more easily carried over short distances than long distances, infected places outside a district therefore are not so likely to be a source of infection to that district as infected places within the district itself. It is very important therefore to locate such villages and endeavour to imitate nature's method of bringing an epidemic of plague to a close by using every means in our power to destroy the rats in these villages so as to limit as far as possible the opportunities for the spread of infection from them. As I pointed out in the paper I read before the Bombay Medical Congress in 1909 the prevention of the importation of infection can be carried out with the best hope of success at the close of the active plague period, it is then that the disease can most easily be tackled, the forces of nature are with us; yet this is generally the period chosen for holidays and the reduction of the plague staff. These arrangements should as a matter of fact be reversed and the greatest effort made to stamp out the disease during the hot weather.

The large cities may be regarded as permanent centres of infection in which unceasing efforts at eradicating the disease should be maintained, and here the permanent measures I have detailed at length above are most applicable. Temporary centres of infection during the quiescent plague period are also important; they are found in the smaller towns and villages and they vary in position from year to year. A thorough system of intelligence is necessary to mark them down, and they should be attacked by well organised plans of rat destruction, inoculation, and evacuation.

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PART III.

VACCINE LYMPH.

ALL-INDIA SANITARY CONFERENCE,—MADRAS,—NOVEMBER 1912.

VACCINE LYMPH.

Production, Preparation and Preservation.

BY

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It is not possible to consider such points as the production, preparation and preservation of lymph separately from one another. Thus for the production of lymph we require to have lymph which has been already *prepared* and probably also *preserved* for some time and therefore production is not independent of preparation and preservation. At the same time while we recognise this dependence it is convenient to consider our subject in more or less consecutive order. We may postulate ideal cases and treat production of lymph, for example, as if we had at our disposal a perfectly prepared and perfectly preserved vaccinating lymph. In the same way we may under the head preservation take up the consideration of the subject of potency which implies a reference not only to ultimate efficiency, but also to modes of preparation and production.

These explanations will suffice to justify my sub-division of the subject.

I.—PRODUCTION.

I need not describe, except in general terms, the methods used in obtaining the raw product which is made up into vaccine lymph. In its production it is still necessary to use an animal of some sort. The animal itself is inoculated with vaccine lymph. The eruption which results is removed as a whole and made up into the finished product. Many factors are concerned in the development of the all essential eruption.

(1) *Animal used.*—Before the introduction of calf lymph, and indeed for some time after, the material obtained from the human vesicle was that used for the continuance of vaccination. Since the introduction of calf lymph and with it the possibility of preservation in bulk, attempts have been made to obtain lymph from other animals than the cow calf. In the Punjab the buffalo has served the purpose of vaccinifer for many years. The results have been excellent both as regards quality and quantity, although it would be going too far to say that they showed a marked superiority over those given by the cow calf. Chauvean asserts that the horse is the animal most suited for the production of vaccine lymph. Huon of Marseilles concludes strongly in favour of an asinine vaccine—a recommendation originating I believe with Chaumier. He argues (1) that the potency of a bovine vaccine is greatest when the strain used for inoculation is donkey lymph: (2) that on the donkey itself bovine vaccine gives as a rule excellent results, and that still better are obtained where donkey lymph is used to inoculate donkeys: (3) that sub-passage in the cow calf sooner leads to deterioration in quality of the lymph than in the donkey. There seems to be a fairly general consensus of opinion in favour of the use of the animal. The point would be worth investigating in India.

I have tried the goat as a vaccinifer but with ill success. The vesicles which developed were apt to contain much fluid and very rapidly became purulent. The rabbit has been greatly used after the recommendation of Calmette, but owing to its size, it has been adopted rather as an animal for regenerating lymph than for its production on a manufacturing scale.

(2) *Race and age* of animal.—Many vaccine institutes lay very special stress on the race of cow calf used. This, no doubt, experience has shown to be important. As far as I have observed, little or no perceptible difference exists between the results obtained with the cow calf of the Punjab and the buffalo calf. The age of the animal might be expected to lead to some difference in the yield of lymph. But I have found that the use of large sized buffalo calves of two years old proved an advantage rather than otherwise because of the greater vaccinable surface afforded. Dr. Guentcheff in Sofia uses heifers of any age from one to four years. Of course the older an animal is the less manageable does he become. The monetary value too of the animal for other purposes than vaccination grows with age. This question of the cost of the vaccinifer is an important one in relation to the cost of production of lymph. The cost will—after initial expenditure on purchase and maintenance—be dependent on the method of disposal of animals which is adopted.

(3) *Disposal of animals.*—In the days of calf-to-arm vaccination, when the villager's calf was impressed for the purpose, only a very small number of insertions was made on the animal and a correspondingly small number of persons treated from it. The return of the calf to the owner involved him in no loss, nor did the operation upon the animal cause it much inconvenience. With the introduction of preserved lymph it became advisable to obtain as much material as possible from the vaccinifer. The operation of removal of lymph uuder these circumstances takes time to recover from and I think the best and kindest plan then is to have the animal slaughtered. Is the flesh of such an animal fit for food? If it is saleable, then the price of sale will reduce materially the cost of purchase and maintenance and so reduce that of production. I had no difficulty in Lahore in disposing of these vaccinated calves. In Brussels, Henseval and Convent likewise find no difficulty in disposing of calves and rabbits used for the production of lymph—these being used for food.

(4) *Quantity of lymph* obtainable from a vaccinifer.—Some hold the view that a calf may be over-vaccinated. Such was the opinion expressed to me by Dr. Blaxall at the Local Government Board Station. Others apparently attach no importance to the view that the potency of lymph may be affected by attempts at increasing the productiveness of the calf. I found, on taking over the appointment of Deputy Sanitary Commissioner, Punjab, that the yield from vaccinifers was very low indeed. This was due mainly to the fact that there was no urgent necessity for a large yield. Preserved lymph at that time was not widely used as such. Its chief use was to start the vaccination of cow calves at the beginning of the vaccination season—the continuance of this being by means of the calf-to-arm method. By degrees the yield was increased to 5, 10 and 15 grammes, whilst in the hands of Major Forster, I. M. S., it reached as much as 40 grammes from a good sized buffalo calf. Now 40 grammes of crude lymph has added to it 4 parts of 50 per cent. glycerine, thus giving approximately, from a single buffalo calf about 200 grammes of final product. I found that with care a vaccinator could vaccinate 40 persons with 4 insertions each from one gramme. Thus I calculate that from one buffalo calf some 8,000 persons may be inoculated. The amount of crude lymph obtained, and on which this calculation is based, is evidently nowhere near the outside limit of possible production by a vaccinifer. With special methods of vaccination and using heifers of two to three years of age, Chaumier has been able to obtain an average yield of 700 grammes of crude lymph and a maximum of 900 grammes. He calculates on an ability to vaccinate 100 persons from one gramme of vaccine, and thus at a dilution of 4 parts of 50 per cent. glycerine to one of crude lymph it becomes possible to inoculate (number of insertions not stated) 350,000 and even 450,000 persons from one animal. At such rates it would not take many calves to supply vaccine for a province.

(5) *Site and extent of inoculation.*—The site most usually chosen is the abdomen with extension in some cases to the inner aspect of the thighs. The extent of surface utilised seems however to depend on our idea of what the animal can stand and on such ideas as we may hold of over-vaccination. Chaumier, whose results in respect of quantity of lymph obtainable I have given above, has vaccinated since 1895 the entire trunk of the animal and his scarifications are so close together that we may call his method one of sheet inoculation. A happy

medium between the usual method followed in India and this method would result in greater production. I used what I called a method of feathering of primary incisions to increase the area of vesiculation.

(6) *Season of inoculation.*—The effect of high temperature upon the potency of vaccine lymph will be specially considered in its proper place, but temperature has likewise or appears to have an effect upon the satisfactoriness of development of the vesicle upon the vaccinifer. Here in Madras, I think you are well acquainted with the difficulty of obtaining good or sufficient material from your calves in the hot weather. In the Punjab the vaccine depôt is removed to the hills in the hot weather and the results are naturally as satisfactory during this season as in the plains during the cold weather. Chalybäus in Dresden found that the vaccine produced during the cold season showed itself more powerful and was more durable than the vaccine of the warm season. Vaccination of calves during the months of June, July and August was given up by him. The temperature of the stables was not allowed at any time to go beyond 16—18° C (60—64° F) because a more elevated temperature accelerated too much the development of the vesicles and diminished the value of the pulp. The effect of light, apart from temperature, may be a factor influencing the production of lymph. The production would seem to be better in dark stables. Humidity of the atmosphere almost certainly has an influence upon the size of vesicles produced and so upon quantity of lymph obtainable.

(7) *Maturation Interval.*—This varies a little with different animals, but the only rule to be observed as regards this is that the lymph should be removed when vesiculation is complete and before pustulation sets in. On buffalo and cow calves the limits of time to removal are 5 to 7 days. The maturation interval appears to be shortened with prevalence of high temperature and diminution of humidity in the atmosphere.

II.—Preparation.

The subject heading of this section is taken to include all matters connected with the preparation of the final product.

(1) *Manufacture* of lymph from animal pulp.—In the days of arm-to-arm vaccination, desiccation of vesicular fluid upon "points" served to provide material for the continuance of vaccination for occasions when children might not be present to permit of the actual direct transference of the contagion. The method is one of preservation of lymph, but has also been used for the preparation of lymph for transmission. It does not, however, seem to me to offer any great advantages over the other methods of preparation in which fluid or semi-fluid substances are used to dilute the pulp. Of these we may consider especially lanolin, vaselin, and glycerin.—

(*a*) Lanolin. This medium was introduced by Colonel King, I.M.S. It gave a lymph of convenient consistency. Lanolin lymph served a very useful purpose and still continues to do so. It enabled lymph to be diluted without interfering with its potency and so greatly increased the quantity obtainable from the calf.

(*b*) Vaselin was introduced as a medium by Colonel Bamber, I.M.S. As a diluent for crude lymph it did great service particularly in bridging over the interval—the hot weather—between vaccination seasons. It was also used to some extent for general vaccination purposes. Vaselin and lanolin, while both of them sterile substances as obtained in commerce, exercise neither of them any inhibitory influence upon the bacterial content of vesicular pulp.

(*c*) Glycerin. This is extensively used now as a diluent of calf lymph all over the world. Its use for the purpose was first advocated by Dr. Monckton Copeman. The great advantage of glycerin is that it exercises a distinct influence on the bacterial content of the mixture without adversely influencing its potency as vaccine. The dilution which crude lymph may undergo whilst still remaining satisfactory, does not seem to have been worked out very definitely. The point is an important one because in times of stress such as

those of epidemic visitation, it might be of great use to know exactly what dilution the lymph held in stock might be subjected to, that is supposing there was insufficient time to produce a new supply of lymph for issue. But much more important than dilution of lymph pulp is the question of purification.

(2) *Purification.*—The crude material from which vaccine is prepared consists of the scrapings obtained in removal of the cutaneous eruption developed on the vaccinifer. Naturally these scrapings contain innumerable skin organisms. It is these skin organisms which seem to give rise to the inflammatory reactions so often seen in the course of vaccination. To my mind such reactions are not evidence of activity of lymph but of its degree of contamination with extraneous organisms. My experience with the two forms of lymph which were used during the time that I had control of the special staff of vaccinators in the Punjab, confirm that opinion. Some of the lesions produced by vaselin lymph—a lymph rich in extraneous organisms—were very severe. Crateriform ulcerations due to sloughing out of the centre of an insertion were common. I saw none such from the use of glycerinated lymph and this lymph is almost entirely free from the presence of bacteria. The association of absence of bacteria and absence of inflammatory reaction would appear to be a true one. Of the process used in the manufacture of vaccine, those in which vaselin and lanolin are used afford no apparent purification of the product. Desiccation of lymph undoubtedly brings about a certain reduction in the bacterial content of lymph, but naturally effects no reduction of such organisms as resist desiccation. With the help of glycerin on the other hand an almost complete purification of vesicular pulp can be brought about. The special point to be attended to in regard to this purification is, that it shall not be carried too far, for unfortunately glycerin will in time attack the true vaccine virus. The process too takes time and requires also a certain temperature. The higher the temperature the quicker is the purification effected, but at the same time the greater is the liability to destruction of the vaccine virus. At low temperatures the purification is extremely slow. Thus, with glycerin as a purifying agent, we are unable to use the prepared vaccine immediately because of its impurity and we are unable to preserve it under cold storage conditions because that itself interferes with the purification. And so we are liable to find that our glycerinated lymph at the time of use is impure or avirulent. It is difficult to steer satisfactorily between this Scylla and Charybdis. A solution of the difficulty, however, seems to have been found in using a rapid purifier of lymph which can be got rid of after its purpose has been effected and which can then be replaced by glycerin. The glycerin is then used only as a dilutent and preservative. Such lymph may be placed without fear of retarding purification under cold storage conditions. The rapid purifier which possesses the property of leaving the vaccine virus unaffected by its action, and at the same time being removable from the mixture, is chloroform. The use of this re-agent was introduced by Doctor Alan Green. I tried it experimentally in the Punjab at the instance of the then Sanitary Commissioner, Colonel Bamber, I.M.S., and obtained satisfactory results. The procedure was then adopted as a routine measure. The advantages in using chloroform which seem to me paramount are that :—

(*a*) There is no delay in the utilisation of the lymph which can be tried on the very day of its manufacture.

(*b*) The lymph after its rapid purification and after the removal of the chloroform can be kept at a low temperature (in chest or refrigerator) in glycerin admixture. Of course care must be taken to remove all traces of chloroform. The procedure consists in the passage through an ordinary lymph mixture of air saturated by previous passage through pure chloroform. One to two hours passage is sufficient for reasonable purification. One of the difficulties to be overcome in this process is the prevention of formation of an excessive amount of froth. This can be done by making the delivery tube of wide bore. The chloroform is got rid of from the mixture by detaching the intervening chloroform bottle and allowing sterile air to bubble through in place of the air laden with the chloroform. Voigt of Hamburg remarks with regard to chloroform lymph that he has only observed bad results

from its use and he is very astonished to find in the sanitary reports of Western India that this vaccine gave in the Punjab in the years 1906-1908 positive results in upwards of 99 per cent of cases. He goes on to say that as no mention of the use of this vaccine occurs in subsequent reports the conclusion is that it is no longer used. But Lieutenant-Colonel Wilkinson, the present Sanitary Commissioner in the Punjab, tells me that chloroformed vaccine has been used and is still used in the Punjab. The results which I obtained in the years 1904-1905 were evidently still obtained in 1906-1908 and are presumably still obtained. Voigt looks at the question from the point of view of despatch of vaccine to Colonies situated in the tropics. According to him both desiccated and lanolinated vaccine withstand a high temperature better than glycerinated vaccine, but lanoninated vaccine gives much better results than desiccated vaccine. My own experiments (comparatively few in number) instituting a comparison between lanolin lymph and glycerin lymph, both subjected for a short period to the temperature of the incubator (37°C), did not bear out the idea of the superiority of lanolinated lymph over glycerinated lymph. Doctor Martin, however, the Director of the Lister Institute, in a conversation also maintained the superior keeping properties of lanolin lymph. We may perhaps conclude by saying that further experimentation on the subject is desirable. It might for instance be preferable to mix lanolin with chloroformed lymph rather than glycerin.

(3) *Degeneration and Regeneration* of lymph.—This subject may be discussed here as it involves the consideration to some extent of production, preparation and preservation, the three main sections of this paper. Now we may take degeneration of stock lymph to be a fact. It comes into the experience, we may say, of all institutes and measures have to be taken for rejuvenescence. When I visited the vaccine lymph station at Halle in Germany, I asked the Director what he did when his stock lymph degenerated, and the answer was, I get fresh lymph from another institute. This is one way of getting over the difficulty, but gives no explanation of how lymph should be regenerated. Chalybäus experimenting to determine how long sub-passage of lymph in vaccinifers might continue without degeneration setting in, succeeded in carrying the same strain of vaccine up to the 20th and even to the 22nd and 30th generations on cow calves. Nevertheless he concludes as to the necessity of regeneration after the third sub-passage, either through the agency of the human subject or by using some other animal than the calf (horse, donkey, goat, sheep or rabbit) or by obtaining a new strain of vaccine from a variolous case—a variola vaccine. In the Punjab I found that alternation from cow calf to buffalo calf, and *vice versa*, served to keep the stock lymph virulent. Evidently this alternation of animals is useful for the regeneration of lymph. It is a method which was in use by vaccinators before the introduction of glycerinated lymph. Passage through the human subject was the mode of operation. A number of children with well developed vesicles are collected together and vaccination done directly from arm to calf. The result of this inoculation is not very productive, but when the small amount of material so obtained is transferred to another calf, the eruption is less scanty and the lymph becomes regenerated. The rabbit, although a small animal, can be satisfactorily used for regeneration of lymph. But what is the cause of the degeneration?

It may be due in the first place to the continuous use of one and the same kind of vaccinifer. Most probably more than one cause is concerned in the matter. Vaccine lymph does not consist of pure vaccine virus. There is in it a plentiful admixture of extraneous organisms. With continued sub-passages of these extraneous organisms, they may gain virulence, whereas the virulence of the vaccinia virus may remain fixed. But with this increase in virulence of extraneous organisms we may have overgrowth, an overpowering of the specific virus and degeneration, not attributable to the vaccinifer used but to the contamination of the original lymph. Transfer this lymph before it is too late to an animal specially susceptible to vaccinia, and as the extraneous organisms are

transferred to foreign ground they may be rendered comparatively innocuous and the vaccine virus gain ground over them—become regenerated. Such at any rate is a possible explanation.

(4) *Stock Lymph and Issue Lymph.*—By means of these terms we differentiate between the lymph which is used for the vaccination of animals (stock lymph) and that which is issued for use in the protection of human beings against small-pox. As the production of issue lymph depends for its potency on that of the stock lymph, it is but common sense to say that the utmost care must be taken that the stock lymph shall be the best lymph available. For stock lymph we should take the best vesicles from the best eruption obtained. The lymph which has proved itself good stock should on no account be issued, but kept under optimum conditions for preservation. With such precautions the danger of unexpected degeneration will be warded off.

III.—Preservation.

The preservation of the potency of any given lymph will depend to a considerable extent on the measures adopted for its production and preservation—the animal used, the character of the eruption, the diluent and preservative added to the crude material, the season of the year at which taken, and so on. These have been already considered. What we now may discuss are the best means of keeping potent the manufactured lymph, the tests of potency and the methods of issue best calculated to preserve potency.

(1) *Maintenance of potency.*—A weak lymph, that is to say one which is derived from an unsatisfactory eruption on the calf, will not preserve well. A lymph likewise which has been taken after pustulation has set in is not likely to remain potent long. But apart from errors of production and preparation, the keeping qualities of a good lymph depend largely on its after treatment. One consideration alone seems all important in this connection and that is the temperature at which it is preserved. This must be as low as possible—refrigeration temperature indeed. To my mind this necessity is what gives the advantage to the method of purification by chloroform. There is no waiting for purification before the lymph may be passed into the cold storage chamber or ice chest. Stock lymph which we have insisted must always be the specially chosen lymph, should only come out of the refrigeration chamber at the time of use. Issue lymph unfortunately has to encounter vicissitudes of temperature before its utilisation. In Lahore during the cold weather, so cold that ice making manufactories shut down, I still found it necessary to procure ice for the preservation of the lymph. It had to be procured from as far away as Karachi. If the chloroform process is not used, then it should be laid down that the lymph produced at a station be put under refrigerating conditions as soon as purification is reasonably far advanced. The degree of purification necessary refers perhaps as much to the type of organism present as to the numbers. Lymph should always be certified free from streptococci. In the matter of inflammatory reaction produced by use, staphylococci are by no means as important as the streptococci.

Conservation of vaccine is a necessity for all institutes and the longer that conservation can be satisfactorily carried the better the position of the institute as regards continuity of supply and preparedness for epidemics. Lymph must be able to be kept, if demands are to be met promptly. According to Chaumier who repeated and confirmed experiments by Blaxall and Fremlin, a good vaccine lymph placed in a refrigerating chamber at—10° C, will keep for a year, two years, or more. The attainment of such a low temperature, however, is by no means easy, but it may be laid down that the preservation temperature should not be allowed to rise above + 10°C.

(2) *Standardisation of Lymph.*—Vaccine lymph is very liable to deteriorate, and it is necessary to have some standard by which to gauge its potency at the time of preparation and again, if possible, before issue. An estimate of potency made at the time of preparation will give some idea of the length of time to which such lymph may be kept. The test before issue is the test that it *has* kept.

(*a*) Test at preparation. This may consist—as has been used by Major Forster, I. M. S.—in marking off on the prepared surface of the

vaccinifer of a definite area with silver nitrate and inoculating this area in the same way as the remainder. I should recommend as being still more precise the use of a weighed quantity of the stock lymph for this area. The product of this area is weighed and the weight is an indication of the satisfactoriness of the result. The character of the vesiculation may also afford indication of potency. Another test—that proposed by Calmette and Guerin—consists in inoculating rabbits with serial dilutions of the lymph to be tested and observing in what dilution vesicles cease to be produced.

(*b*) Test before issue. This is best carried out at a vaccinating station. Such a station should be attached if possible to the institute itself. The number of insertions which prove successful is noted as is also the character of the vesiculation. With linear scarification the production of no vesicles or only isolidated vesicles on the line is indicative of a poor lymph. A fimbriated margin shows a condition intermediate between poor lymph and that produced by the best lymph, namely a continuous unindented margin. All results obtained with issue lymph should be capable of being traced back to its source, and so by comparison of these results with those obtained with the same material in other hands, an idea will be obtained as to where a fault, if any, lies—with the lymph or with the vaccinator. According to Chaumier the quality of a vaccine subjected to test by means of the character of the vesiculation is best estimated on the 3rd day. He considers that often the ultimate results of good, bad and indifferent vaccines may be very much the same.

(3) *Issue of lymph.*—All possible provision may have been made for the preservation of lymph while retained in the institute, but this may be nullified by its treatment after despatch. After issue the temperature conditions are not so easily controlable as before it. It is self-evident, however, that everything should be done in issuing vaccine lymph to prevent the adverse influence of high temperature. Its issue, as I believe is done in some parts of India, in thermos flasks is a step in this direction. Its utilisation should on no account be delayed as this increases greatly the chances of deterioration. I am doubtful whether any lymph used later than a fortnight from the date of its despatch will prove consistently satisfactory. Another circumstance which has a great influence on success or non-success of vaccination is the time of year at which the operations are conducted. Be the issue lymph good or bad and be the arrangements for transport to the vaccinator good or bad, the results obtained during the hot weather will never compare with those of the cold weather. Therefore all vaccination should be conducted during the cold weather months. In the Punjab vaccination is carried out during the months of November, December, January, February and early March. The results are certainly satisfactory.

The main points of this survey may be set down as follows :—

(1) High temperature with attendant dryness of the atmosphere is inimical to the production of satisfactory vesicles by vaccinifers.

(2) Stock lymph must always be the best lymph obtainable and must be most carefully protected from influences leading to deterioration.

(3) Degeneration in the lymph supply appears to set in if one and the same type of vaccinifer be used.

(4) Refrigeration of a continuous type is essential to the preservation of both stock and issue lymph.

(5) Lymph which has been issued must be preserved from the action of high temperature and used as soon as possible.

(6) Vaccination operations should be confined to the cold season of the year.

PART IV.

DRANCONTIASIS.

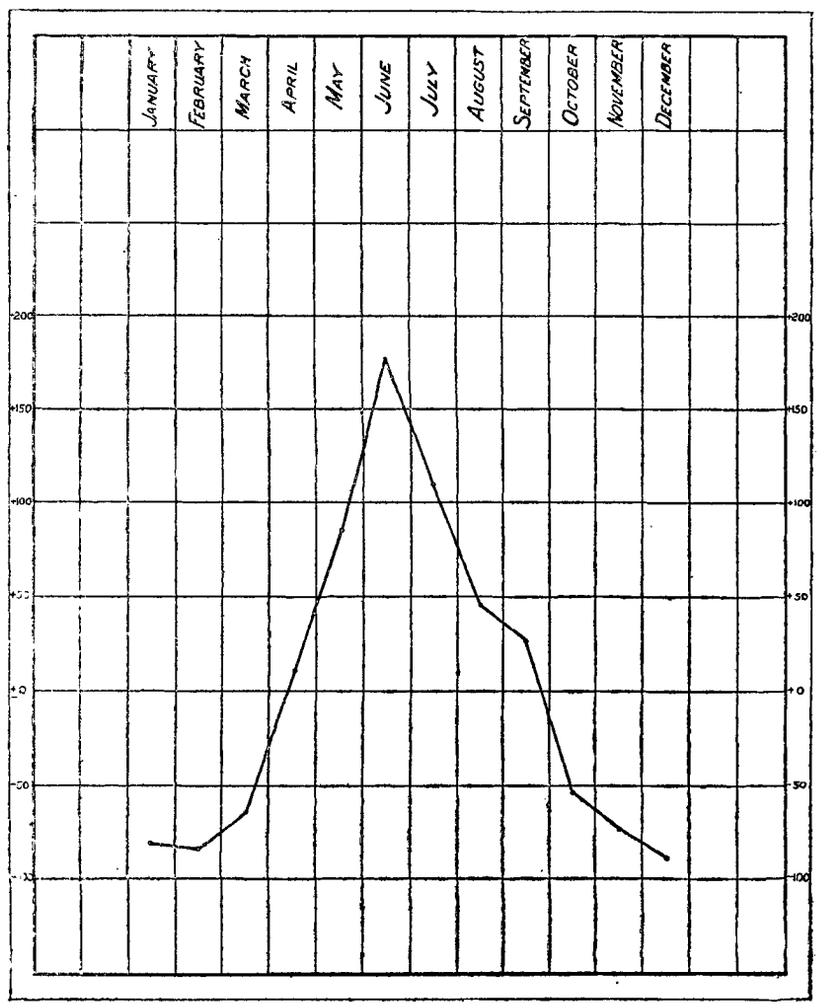
DRACONTIASIS

BY

DR. D. A. TURKHUD, M.B., C.M. (Edin.),

BOMBAY BACTERIOLOGICAL LABORATORY.

The amount of suffering and physical infirmity caused by guinea-worm in certain districts in the Bombay Presidency is very great and not often fully realized. In some villages a very large percentage of the inhabitants are more or less completely incapacitated from work for certain months in every year. In the famine camps in the Panch Mahals District, for instance, the Collector reported 2,000 cases of this disease in the month of March 1912, and this was practically the only illness among the people.



The seasonal prevalence of the disease is well seen in the above chart prepared from the figures showing the admissions for guinea-worm for the past 5 years in all the dispensaries of the Panch Mahals District; the chart shows for each month, the percentage figures above and below the mean. The seasonal outbreak appears to last from April to September with the maximum manifestation of the disease in the month of June.

The disease is caused by a filaria—filaria medinensis—but its life cycle is not yet thoroughly known.

The prevailing theory as regards the life cycle of the worm is as follows:—

Only the adult female worm is known. When she comes to the surface of the skin in a human being she is already distended with fully developed larvæ, and upon her coming into contact with water she immediately discharges into it a very large number of her larvæ. The larvæ then find their way into the body cavity of cyclops which are small crustaceans and very common inhabitants of all wells and ponds. Within the cyclops the larvæ undergo certain changes, and when such infected cyclops are taken into the human stomach by means of drinking water, the gastric juice kills the cyclops and liberates the contained larvæ. The further life history of the worm is not known, but about a year afterwards fully developed adult female worms make their appearance on the surface of some part of the human body, generally the legs.

The life cycle of the worm may be represented as follows :—

Host and duration of the worm within the host.	*Life cycle of the filaria.*
Man (Duration ?)	The female filaria in the connective tissues of the host, chiefly in the extremities.
	The adult worm full of living embryos or larvæ reaches the skin.
(1 to 100 hours ?)	Larvæ escape into water.
Cyclops (six weeks ?)	Larvæ in the interior of cyclops. They undergo certain changes within the host.
Man (10 to 12 months).	Infected cyclops swallowed by human beings, with drinking water.
	The larvæ escape from the cyclops into the human stomach.
	? ? ?
	? ? ?
	Female guinea-worms find their way into the connective tissues of the host, chiefly the extremities.
	The adult filaria full of living larvæ reaches the skin of the host.

It will be seen that some of the important points in the life cycle of the filaria are still not known; how and where the female becomes fecundated and what course the worm follows after being swallowed until it reaches the surface of the skin in the human being—these facts have yet to be worked out.

The fluid of the blister made by the worm in its attempt to reach the surface of the skin is found to be full of living embryos in an active condition. Whether these embryos or larvæ are capable of travelling along the channel in the tissues made by the mother worm and thus re-infect the human host is not known. Such direct method of infection appears to be very doubtful; but experiments are being made on monkeys by introducing the living larvæ subcutaneously.

As regards the manner in which the larvæ, discharged into water, enter the bodies of the cyclops, there is a good deal of controversy. Fedschenko believed that the penetration is effected through the integuments of the cyclops and not per os, and Manson and Blanchard have confirmed these experiments.

Wenyon states that the embryos gain entrance to the body of a cyclops by piercing it with their sharp tails.

Leiper maintains that the mode of entry of the embryo is not through the integument of the cyclops, but by way of its intestine (British Medical Journal, January 19th, 1907).

The observations carried out at Parel Laboratory showed that the larvæ are actually swallowed by the cyclops per os. These observations were confirmed by Captains Gloster, White, Kunhardt and Stevenson working at the Laboratory, and can be repeated by anyone by taking the precaution of previously starving for a day or so the cyclops to be experimented with by placing them either in clear tap water or in normal saline solution. A hungry cyclops will then be seen to swallow the larvæ in rapid succession and the larvæ can be traced within the cyclops' stomach which will be seen more or less firmly contracted upon them. Within half an hour, however, the larvæ will be found to have gained entrance to the body cavity of the cyclops, probably through a tear in the stomach wall.

The changes which the larvæ undergo within the cyclops will be described in detail at a subsequent date; these are evidenced in a shortening and thickening of the posterior extremity, the loss of cuticle and certain cellular changes within the body.

The longest period for which the embryos were kept alive at the Parel Laboratory within a cyclops was 53 days and at the end of that period the embryos still showed sluggish movements inside their hosts.

A number of monkeys have been fed by mouth on cyclops infected for periods varying from 6 to 53 days. The accompanying table shows the differential leucocyte count of two of these monkeys. The first monkey fed on cyclops infected only 6 days previous to the date of feeding showed no change among the leucocytes three months afterwards.

Monkey No.	Date of feeding.	No. of infected cyclops given.	No. of guinea-worm larvæ given.	Duration of infection in cyclops.	Leucocyte count	
					Date.	Date.
					20-7-1912.	29-9-1912.
1	20-6-1912	10	29	6 days	Polynuclears ... 36%	40%
					Eosinophiles ... 4%	6%
					Lymphocytes ... 23%	30%
					Large mononuclears 37%	24%
2	24-6-1912	8	18	10 days	Polynuclears ... 49%	44%
					Eosinophiles ... 4%	23%
					Lymphocytes ... 25%	24%
					Large mononuclears 22%	9%

The second monkey fed on cyclops carrying infection for 10 days showed marked eosinophilia after three months. A careful *post mortem* examination of this monkey was made on 2nd October 1912, but no worms were detected either in the internal organs or in the cellular tissue around the viscera.

In our experiments at Parel, guinea-worm larvæ were also found inside a Stegomyia larva, but whether they undergo any further development in this host is still to be ascertained.

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PART V.

SLEEPING SICKNESS.

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SLEEPING SICKNESS

A SUMMARY OF THE WORK DONE BY THE SLEEPING SICKNESS COMMISSION, 1908—1910.

By

CAPTAIN F. P. MACKIE, I.M.S.

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The Sleeping Sickness Commission of the Royal Society, 1908-1910.

Constitution.

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The following is a short account of the present state of our knowledge of Sleeping Sickness and has special reference to the work of the above Commission. If in these pages any statements are found which seem to differ from those made in the official publication of the Commission the responsibility for them is mine alone.

In the compilation of this report I have had frequent resort to the Bulletin of the Sleeping Sickness Bureau, to the Editor of which I tender my thanks.

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SLEEPING SICKNESS.

Sleeping Sickness is a disease affecting man and is caused by a blood parasite, the *Trypanosoma Gambiense* (Dutton), which is transmitted by the bite of a species of Tsetse fly known as *Glossina Palpalis* (*Rob-Dev.*). The disease is confined to the tropical and subtropical regions of Africa, but it is slowly spreading along the line of human communication.

Distribution.—The clinical symptoms of this disease were recognised more than a century ago on the West Coast of Africa, and about that time the disease made its way into the New World amongst the negroes who were carried there from Africa as slaves.

In 1901 Forde and Dutton, who were working in the Gambia country, found a trypanosome in the blood of a person suffering from fever and the parasite was called "*Trypanosoma Gambiense*" and the disease "Trypanosome fever."

It was not till Castellani discovered the same parasite in the blood of persons suffering from Sleeping Sickness in Uganda and until the significance of his discovery was emphasised by Bruce and Nabarro that the relationship between trypanosome fever and Sleeping Sickness was fully recognised.

By the labours of Bruce and Nabarro and of Greig of the Indian Medical Service and of Gray and the lamented Lieutenant Tulloch, of the Royal Army Medical Corps, the true relationship of the *Trypanosoma Gambiense* to Sleeping Sickness was established and the parasite was by these observers proved to be transmitted by a species of tsetse fly.

Since then Sleeping Sickness has been found to be a much more widespread disease than was formerly supposed. It appears to have spread from West Africa throughout the basin of the Congo, thence into Uganda where it has caused a terrible mortality.

Since then it has spread southwards into German East Africa and into Nyassaland; northwards into the Lado enclave and into the lower Soudan. Recently there have been alarming reports that it is spreading southwards into Rhodesia, so that altogether the increased extent of its distribution is giving rise to grave concern.

The problem concerned with the disease may be discussed under four headings:—

I.—The disease in man.

II.—The parasite which causes it.

III.—The fly which carries the disease.

IV.—The reservoir of infection.

I.—The Disease in Man.

The present Commission did not specially concern itself with this aspect of the question, but for the sake of completeness a few words must be said about the clinical symptoms.

I.—SYMPTOMS OF SLEEPING SICKNESS IN MAN.

The incubation period is somewhat uncertain but is probably a matter of days or of a week or two at the most. We found that the incubation period in monkeys was almost invariably under ten days and nearly always either six or seven days.

The blood of the monkeys was infective two or three days before trypanosomes were first seen in their blood by microscopic examination.

The first stage of the disease is febrile and is generally put down to malaria. The pyrexia is intermittent and is probably associated with the recurrent appearance of the parasites in the blood. These febrile attacks may recur for months or even for years without involvement of the nervous system and the patient becomes anæmic and debilitated.

Rashes occur during this stage and are either erythematous or vesiculo-papular. Occasionally there are painful nodular swellings or plaques from which the trypanosomes may be recovered. They are apparently similar to those seen in horses suffering from dourine, a disease due to *Trypanosoma Equiperdum*.

Dermatographia, a form of skin affection generally ascribed to vaso-motor disturbance, is not uncommon. These rashes, though they may be of great help in diagnosing Sleeping Sickness in fair-skinned people, are difficult to recognise in dark-skinned races.

A sign first described by Kerandal and known by his name is that of deep hyperæsthesia. When the soft parts, such as the calves of the legs, are pinched or squeezed, acute pain is felt. The sign resembles that commonly found in some forms of neuritis and probably is due to a similar cause.

The most important diagnostic sign is the enlargement of lymphatic glands, particularly those in the posterior triangles of the neck. The glands are soft and elastic aud remain discreet, though they may become large enough to form an obvious swelling in the neck. This diagnostic sign is the most easily recognised of all signs and its value was greatly enhanced by the discovery made by Captain Greig of the Indian Medical Service and Captain A. C. H. Gray of the Royal Army Medical Corps, who found that trypanosomes could readily be found by puncturing the glands in a very large proportion of cases. (97 4% Koch.)

The characteristic signs of the earlier stages of Sleeping Sickness are:— (1) Pyrexia (with or without trypanosomes in the blood stream. (2) Kerandal's sign (deep hyperæsthesia). (3) Certain rashes. (4) Cervical buboes (containing *Trypanosoma Gambiense*).

By means of these signs the disease can be recognised in quite an early stage.

The second or cerebral stage is the one which gives to the disease the name of Sleeping Sickness. The earliest sign is said to be a change in disposition and a loss of mental and moral tone. The patient becomes dull and apathetic

and often dirty in his habits, showing at the same time a disinclination to work or to exert himself at all and a tendency towards somnolence on being left to himself.

Fine tremors occur in the fingers and extend to the limbs and head; incoordination and even Rombergs symptom is met with and rigidity sets in in certain muscles.

Headache is common throughout the stage and occasionally hyperæsthesia or neuralgias. The Argyl-Robertson eye symptom is stated not to occur and Babinski's sign is absent.

The other vital organs are not generally affected, but as the disease progresses the patient becomes more and more somnolent and eventually passes into a condition of coma, in which he may linger for days or weeks before death finally occurs. There are several features in the above condition which will remind the clinician of the late stages of general paralysis of the insane, and when one remembers the similarity of the underlying histo-pathological condition the resemblance of the two diseases is not surprising.

II.—THE PARASITE OF SLEEPING SICKNESS.

The *Trypanosoma Gambiense* is a protozoon belonging to the class *Mastigophora.*

In length it is from 17-28 microns and in breadth about 2 microns. When observed in the blood of man or experimental animals it does not vary much in size, but when undergoing development in the interior of the tsetse fly very great differences in size and shape may be observed.

In structure it resembles other typical trypanosomes in possessing a macro-nucleus situated medially, a micro-nucleus near the posterior extremity and a well-defined undulating membrane which commences at the posterior extremity in the neighbourhood of the small nucleus and passing along the whole length of the body is continued anteriorly as a free flagellum.

The *Trypanosoma Gambiense* is an obligatory parasite, being only known to exist in the tissues of human beings suffering from Sleeping Sickness, in the tissues of certain of the lower animals and in infected tsetse flies.

Some observers claim to have grown it in cultures on Novy's blood agar medium. Multiplication takes place by longitudinal division, the process beginning in the region of the micro-nucleus and extending forwards to result in the formation of two perfect daughter cells. Sexual dimorphism is suspected but has not been proved, and sexual conjugation has never been witnessed.

The changes which the parasite undergoes in the body of the tsetse fly will be described later.

III.—THE FLY WHICH CARRIES THE DISEASE.

The tsetse fly *Glossina Palpolis* (Robinean-Desvoidy) is the insect responsible for the transmission of Sleeping Sickness.

It belongs to the Genus *Muscidæ* of Dipterous insects.

It is a dark insect about the size of a "blue bottle" fly and inhabits a narrow belt of wooded country on the shores of certain African lakes and the watercourses of rivers.

It is never found very far from water and is restricted to a narrow belt near to the water's edge and generally within a few hundred yards.

It has a quick, almost noiseless flight, and settles suddenly on its victim, driving its long powerful proboscis deep into the skin and filling itself with blood in about half a minute. The puncture is generally quite painless but in some people is followed by mild irritation and by a weal or lump.

The fly, which is thin and flat when empty, distends its abdomen to the shape and size of a small red currant after a feed of blood and then flies heavily away to digest its meal.

Although it is curiously local in its distribution it sometimes travels on the bodies of men or animals or in canoes from one part of the shore to another, and even may be found a mile or two inland when accompanying caravans.

When the local conditions are favourable the flies swarm in large numbers and are exceedingly voracious, even biting Europeans through thick clothes. The *Glossina* are remarkable in that they produce a single living larva at a birth instead of laying eggs as is the manner of most flies.

The larva, which is a small maggot-like creature, wriggles actively, and by this means burrows into the superficial layers of soft loamy sand by the water's edge, and in a few minutes becomes darker and motionless and becomes transformed into the pupa or chrysalis stage.

The perfect insect emerges after a time varying according to the locality and climatic condition but probably in about six weeks.

Tsetse flies only bite in the daytime and preferably in the heat of the day.

The duration of their lives is certainly over 96 days, for the Commission kept some in captivity for that length of time, so that under favourable conditions in nature it is probably longer.

THE FOOD OF THE TSETSE FLY.

The natural food of Glossina Palpalis is a question which has received a great deal of attention.

It is admitted on all hands that the tsetse fly is a pure blood sucker and there is no evidence whatever to show that it ever lives on vegetable juices. In its natural haunts near the water's edge on the fringe of vast forests there is plenty of animal life available.

Professor Koch was of opinion that the chief source of its blood supply was the crocodile, a statement which was misquoted to the effect that he believed the crocodile to be the source of Sleeping Sickness, which he did not state.

It is true that the tsetse fly will suck the blood of the crocodile just as it will that of almost any creature which contains blood, but there are many places where tsetse flies are found where there are no crocodiles.

In the large majority of the flies caught on the lake shore there is no evidence of food of any sort; this is doubtless because only the hungry and therefore empty flies come out and are caught. Thus out of a series of 120 tsetse flies brought to the Commission's laboratory for dissection, only 27 per cent. contained the remains of blood, and out of this number the corpuscles in 9 per cent. were recognised as being of mammalian origin and 4 per cent non-mammalian. In the remainder the blood was too far digested to enable one to recognise its origin. An experiment on a larger scale was carried out, expressly to determine the point, and full details will be found in paper No. 11 (*vide* Summary, page 23) from which the following extracts are taken. "A journey was made to a small "peninsula hereafter referred to as Crocodile Point."

"The place was distant about two and a half to three hours by canoe from "the Kibanga landing place and lay in a sheltered bay far out of the beaten track "of the canoes which came backwards and forwards between the islands of Kome, "Damba and Buvuma and the weekly market at the Kibanga clearing.

"It was chosen therefore partly because it was isolated from human "influences and also because of the large number of flies which lived there and "the number of crocodile and birds which frequented it.

"When it was first visited a large crocodile was disturbed from where she "was lying outstretched on a spit of sand. The canoe men at once set to work

"and unearthed fifty-eight crocodile's eggs lying in layers about eighteen inches "below the surface over which the 'form' of the parent was clearly defined in "the soil.

"The peninsula was pointed in shape and not more than sixty yards long "and was closed on the land side by the dense wall of forest which fringes the "lake shore.

"It was scattered with light undergrowth and fringed by ambatch trees on "which flocks of divers and cormorant sat with outstretched wings drying in the "sun.

"A barrier of bare rocks and boulders projected on one side into the water "and a small school of hippopotami was generally to be seen floating near and "basking in the sun.

"Many small crocodiles were disturbed from the undergrowth as the point "was explored and various kinds of small land birds frequented the reeds and "shrubs. The soil was sandy loam and shaded by light foliage. Tsetse flies "swarmed . . .

"Subsequent to the experiment the daily catch of Lake shore flies was "obtained from the place and incidentally it may be added that the flies caught "here were found to be regularly infective to monkeys."

Pupæ could be found in situation such as this by scraping the superficial layers of the sandy loam. As many as two thousand were found in a day by three trained pupa seekers.

A rough dissection of all the flies caught on the point was made then and there, and those which contained the remains of a blood meal were smeared on a slide and brought to the laboratory for minute investigation.

The result of the examination of 183 flies caught at Crocodile Point shows that nearly 60 per cent. of flies contained blood chiefly of non-mammalian origin of which reptilian blood was twice as frequent as that of birds.

OTHER INSECT CARRIERS.

This is the place to discuss the very important question as to whether Sleeping Sickness can be carried by any other medium than by the tsetse fly.

Many experiments have been done to prove whether the *Trypanosoma Gambiense* can be transmitted by tsetse flies other than *Glossina Palpalis*. The same has been attempted with bugs, lice, mosquitos and other house parasites.

In a few instances some of these experiments have proved successful, but in the majority they have been inconclusive and what is more important *Sleeping Sickness has never been known to be transmitted amongst human beings in the proved absence of tsetse flies.*

One has to guard against deducing too much from laboratory experiments, for in such cases all the conditions are made most favourable for the success of the experiment and factors which in nature come into counterbalance or modify these conditions are eliminated.

The French Commission described some localised epidemics of Sleeping Sickness on the French Congo with the view of throwing some light on this problem.

They say "It appears that the agent of infection must be an insect biting in the night* when all the members of the family are together, an insect which passing from one to the other may carry the germ to everyone in the immediate surroundings of the patient by its repeated bites. It is mosquitos especially which seem to play the part particularly *Stegomyia* and *Mansonia*, which are so common in the Congo."

* *Glossina Palpalis* only bite during the day.

They sum up as follows :—

" *Glossina Palpalis* is the principal agent of infection. Its presence is absolutely necessary for the propagation of the disease to a distance and for the maintenance of endemicity, but in addition mosquitos and probably other biting insects may be important auxiliares, having a formidable rôle as epidemic agents, exercising their action in the interior of houses and amongst native families, sometimes even throughout villages."

Todd of the Liverpool School of Tropical Medicine referring to the work of the French Commission writes : " Their conclusions entirely harmonise with our own observations."

On the other hand the late Professor Koch wrote regarding a segregation camp where hundreds of patients were collected together :—

> " They were in a country in which *Glossina Palpalis* is altogether wanting but other blood-sucking insects which might be concerned are very abundantly represented ; there occur also in patches *Glossina Fusca Morsitans*. In a district such as this the problem must inevitably be settled whether other insects are able to transmit the *Trypanosoma Gambiense* from sick to healthy men. To decide this very careful observations were made, but not a single case could be discovered in which such transmission had taken place by means of other insects than *Glossina Palpalis*."

There is considerable doubt as to what is the transmitting agent in the form of human trypanosomiasis now spreading in Nyassaland and Rhodesia. The tsetse *Glossina Palpalis* is said to be absent from those regions, but *Glossina Morsitans* is common and *Glossina Fusca* is also found.

It is believed that *Glossina Morsitans* will be incriminated as it has already been proved to be the natural carrier of Nagana (*T. Brucei*) and it is capable of carrying *Trypanosoma Gambiense* by laboratory experiment. Some observers however doubt whether the Rhodesian Trypanosoma is *Trypanosoma Gambiense* at all or that the disease is identical with Sleeping Sickness as ordinarily understood. At present the whole question must remain *sub judice*. The new Commission going out under Bruce will doubtless clear up these points.

There remains one more possible method of transmission, that is either by contact or by sexual intercourse. Koch believed the latter to be a probable mode of spread, and he brought some evidence to prove his contention. This theory has not gained ground and does not accord with the experience of most observers though there are experimental grounds for believing it to be possible.

A recent paper by Hindle (Parasitology, March 1911, page 24) shows that *Trypanosoma Gambiense* can be absorbed through the unbroken mucous membrane either of the alimentary tract or of the female genital passages of the guinea pig.

He did not succeed in transmitting the disease in these animals from infected male to healthy female or *vice versa* by natural sexual intercourse.

Application of infected material to the unbroken skin also gave rise to infection, so it is possible that in man simple contact with infected fluid without a skin lesion might suffice to transmit the disease.

Trypanosomes may, therefore, be transmitted in very varying ways—for the rat trypanosome (*T. Lewisi*) has been proved to be transmitted by the rat flea or by a species of rat louse, the human trypanosome disease of Brazil (*Schizotrypanum Cruzi*) by a reduvial bug whilst dourine, an equine disease, is transmited by sexual intercourse through lesions on the genital organs.

The question has been examined at length because of its vast importance to India and to other hitherto uninfected tropical countries, but this part of the problem will be referred to later in the paper.

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THE DEVELOPMENT OF THE TRYPANOSOME IN THE TSETSE FLY.

As a result of the previous work on Sleeping Sickness it was supposed that the part which the fly played in transmission was a passive one, in fact that it was no more than a mechanical act and that the virus was carried on the proboscis of the fly just as vaccinia is carried on the ivory points of the vaccinator's lancet.

Now we know that the tsetse fly plays a much more important part and that it is in reality a true intermediate host and that *Trypanosome Gambiense* develops in the fly in somewhat the same way, though without such a defined cycle of stages, as the malarial organism does in the interior of the mosquito.

The credit for the discovery belongs to Kleine, who was working in German East Africa and who at the end of 1908 showed that a trypanosome disease could be conveyed by tsetse flies some 50 days after they had fed on an infected animal. We were very shortly afterwards able to confirm this discovery—he had worked with *Glossina Palpalis* and *Trypanosoma Brucei* whilst our experiments were carried out with the same fly and with *Trypanosoma Gambiense.*

The details of the first successful experiments which are recorded in full in paper No. 2 (*vide* Summary, page 23) may be summarised as follows.

The experiment was carried out with flies caught on the Lake shore.

They were first fed once a day on two successive days on an animal whose blood contained *Trypanosoma Gambiense.* Then they were starved for 72 hours to do away with any possibility of "mechanical transference" which was still believed to take place.

For the next five days the flies were fed on a healthy monkey and henceforward changed on to a clean monkey every succeeding five days.

The result was that the first two monkeys remained healthy, but every subsequent monkey on which the flies fed, up to 75 days, developed Sleeping Sickness and died.

If seven days be deducted for the incubation period then the flies became infective 18 days after their first feed on an infected animal.

There is no doubt that all the mischief was done by one fly, because though all the flies which died during the course of the experiment were carefully dissected and at the end all that remained alive were dissected, yet only one fly contained trypanosomes and that fly died on the 75th day. After its death no more monkeys became infected. This fly was swarming with trypanosomes of a Gambiense type and when a minute portion of the alimentary contents of this fly was infected under the skin of a monkey it promptly gave rise to Sleeping Sickness in the monkey.

The salivary glands of the fly contained involution forms of *Trypanosoma Gambiense* and polymorphic forms of the same parasite swarmed in the fore-gut and mid-gut.

After this success a large number of experiments were done on similar lines not only using *Trypanosoma Gambiense* but also *Trypanosoma Pecorum*, *Trypanosoma Vivax* and *Trypanosoma Nanum.* In the first series of these wild or lake shore flies were used and they involved the fallacy that some of the lake shore flies were known to be infected in nature.

When Fraser discovered the true breeding grounds of *Glossina Palpalis* large numbers of pupæ were brought up and hatched out in the laboratory and an abundant stock of "clean" flies was available.

We were able also to ascertain that there was no hereditary infection of tsetse flies so that experiments with laboratory bred flies involved no fallacy.

Full details of these experiments are to be found in paper No. 8, which contains a mass of information of which the bare conclusions were as follows :—

"That *Trypanosoma Gambiense* multiplies in the gut of about one in every 20 *Glossina Palpalis* which have been fed on an infected animal.

"That the flies became infected on an average 34 days after their first feed.

"That a fly may remain infected for 75 days.

"That *Trypanosoma Pecorum*, *Trypanosoma Vivax* and *Trypanosoma Nanum* may also multiply on *Glossina Palpalis* which must be looked upon as a possible carrier in these diseases.

"That multiplication in the tube of the proboscis is characteristic of *Trypanosoma Vivax*."

MECHANICAL TRANSMISSION.

There were still some lingering doubts as to how far this late infectivity of the fly was responsible for the spread of Sleeping Sickness and what part, if any, was played by the hitherto believed method of mechanical transmission.

Some experiments were, therefore, carried out with Laboratory bred flies to prove or disprove the point.

In the first series of experiments a considerable number of flies were used and the infected and the clean animal having been placed side by side the box of flies was transferred rapidly from one to the other until all the flies had fed.

In this way they were not given time to finish their feed on one animal but were constantly changed from one to the other. This method was called "interrupted feeding" and was carried on on twelve successive days. Five experiments of the sort were done and in two of them the experimental animal developed Sleeping Sickness which showed that the disease could be transmitted in this way.

The second method consisted in allowing a variable time to elapse between the feeds and times were chosen from half an hour onwards to 48 hours.

When even such a short interval as half an hour was allowed to elapse between the feed on the infected and the clean monkey, no result was obtained and all the monkeys remained healthy.

The conclusions we arrived at were that :—

(1) The mechanical transmission of Sleeping Sickness by means of *Glossina Palpalis* can take place if the transference of the flies from the infected to the healthy animal is instantaneous—that is by "interrupted feeding."

(2) This mechanical transmission does not take place if an interval of time comes between the feedings.

(3) Mechanical transmission plays a much smaller part, if any, in the spread of Sleeping Sickness than has been supposed.

WHAT HAPPENS TO TRYPANOSOMA GAMBIENSE WHEN IT GETS INTO THE BODY OF THE TSETSE FLY?

Something was said about this question in Paper 2 (see Summary, page 23) which has already been reviewed, but from the time of the first successful experiment there detailed right up till the time the Commission left Africa, an enormous mass of information was collected bearing on the question of morphological change in the parasite of Sleeping Sickness.

It was important to trace the fate of the trypanosoma from the very first hour it was infested by the tsetse fly right up to the time that the fly became infective to monkeys and indeed as long as the infectivity lasted.

It was thought that perhaps the trypanosomes would be seen to differentiate themselves into male, female and in different forms, that sexual union might be witnessed, and that a long cycle of changes as a result of the union might be recognised much as may he seen in the case of the malarial organism in the anopheles mosquito. One must emphasise at the outset that no such changes could be traced and there was no evidence to show that any sexual differentiation took place.

To make the experiment a complete one cages full of tsetse flies were taken and fed on infected monkeys in such a way that a different batch of flies was dissected every day from one day onward to 56 days after the infected feed.

Sometimes it happened that when the flies came to be dissected on a particular day out of the 40 or 50 flies examined none were found to be infected —in this case that particular feeding had to be repeated and so in the end the series was made complete from 1 to 56 days which was as far as the experiment was taken.

When an infected fly was found, a careful dissection was made and all its various organs isolated, carefully washed to exclude contamination, smeared on slides, stained and subsequently studied and drawn under a magnification of 2,000 diameter. In all cases, the organs specially studied were the proboscis, proventriculus, fore-gut, mid-gut, hind-gut, proctodæum and salivary glands.

More than six hundred selected drawings* were studied to form the basis of our conclusions.

When these drawings were placed on a wall the horizontal rows represented pictorially the changes which the trypanosoma underwent, say, in the fore-gut from the 1st to 56th day, whilst those in a vertical direction represented the trypanosomes found in the whole length of one fly on a particular day.

So that, as the original paper adds, "it seems impossible that any important form can have been left out."

One must remember that it is only in a certain proportion of flies—8 per cent. in this series—that any development of trypanosomes takes place. What happens to them in the other 92 per cent of flies is not known, nor why it is that the 8 per cent are alone susceptible to infection.

It was shown not to be a matter of chemical re-action, nor of the presence or absence of intestinal bacteria nor yet ofthe sex of the fly—it must be something more subtle.

The original paper is already so condensed that it is difficult to do more than to emphasise the most important point and to refer those interested in the question to the paper itself.

It was found that in the 92 per cent of the flies referred to, the trypanosomes begin to degenerate a few hours after having been ingested and die out entirely in a few days never to re-appear.

In the remaining 8 per cent the parasites seem to degenerate and become fewer and at a later period to re-appear and undergo enormous multiplication. The appearance under the low power of such a fly is astounding—the whole alimentary canal is distended with a seething mass of trypanosomes and the furthest confines of the dissection fluid quiver with them.

Trypanosoma Gambiense does not undergo any development in the tube of the proboscis (a striking difference to *Trypanosoma Vivax* which in the same tsetse fly seems to infect the proboscis alone and to develop there).

It is in the fore-gut, mid-gut and hind-gut that the chief multiplication takes place, and the type of parasite met here is bewildering in its variety. None of them are typical of those met with in the circulatory blood of the final hosts but there is one form so common as to justify it being looked upon as the "healthy normal developing type in the intestine of the fly."

It is seen at all stages and in all flies. The other trypanosomes are made up of small forms, slender and crithidial forms; resting forms (some of them not

* All the work of Lady Bruce.

unlike *Leishmania*) and giant types—huge bloated masses of protoplasm full of chromatin grannules and waving with flagella.

The behaviour of the trypanosome in the salivary glands merits special attention.

They are not found in these organs until the 25th day after an infected feed; it is only here that the trypanosome reverts to the normal blood type; and before the 25th day the bite of the fly is not infective but it becomes infective synchronously with the appearance of mature trypanosomes in the salivary glands.

To make the above facts more striking one may add that in all the salivary glands from flies which gave a positive result trypanosomes of a mature blood type were found.

There is one important hiatus in the above description. How do the trypanosomes get from the alimentary canal to the salivary glands? The analogy between trypanosomes and malaria is tempting; whilst the malarial parasite is developing in the stomach wall of the mosquito the insect is non-infective, its infectivity coincides with the presence of ripe sporozoites in the salivary glands which find their way there by the rupture of the oocyst into the body cavity of the mosquito. No one has yet witnessed this change in the life history of trypanosome. Very recently however Minchin and Thomson have described the development of the rat trypanosome in the epithelial cells of the alimentary canal of its carrier, the rat flea.

The conclusion arrived at may be summarised as follows :—

1. In the course of the development of *Trypanosoma Gambiense* in *Glossina Palpalis* the proboscis does not become involved as in the case of some other species.
2. A few days after an infected feed the trypanosoma disappear out of the great majority of the flies, but in a small percentage this initial disappearance is followed by a renewed development.
3. After a very short time the flies which have fed on an infected animal become incapable of conveying the infection by their bites and their non-infectivity lasts for some 28 days when a renewed or late infectivity takes place.
4. A fly in which the renewed or late infectivity occurs can remain infective for at least 96 days.
5. An invasion of the salivary glands occurs at the same time as the renewal of infectivity and without this invasion of the salivary glands there can be no infectivity.
6. The type of trypanosoma found in the salivary glands when the fly is infective is similar to the short stumpy form found in vertebrate blood and it is believed that the reversion to the blood type is a *sine quâ non* in the infective process.

Some more experiments of a different class were done to ascertain the question of the return of infectivity of tsetse flies.

They were fed on animals infected with Sleeping Sickness, but instead of being allowed to bite clean animals or being subjected to microscopic examination their infectivity was tested by braying them in a mortar and injecting the resulting intestinal fluid with a hypodermic syringe into healthy monkeys.

A number of these experiments were done and it was proved that the *Trypanosoma Gambiense* retains its virulence for about two days after ingestion by flies when administered in this way.

After two days have elapsed they lose their virulence again, but it returns in a very small proportion of flies in about 22 days.

From this it appears that the virulence of the trypanosome in the fly is lost for just about the same period that the fly is non-infective to animals when allowed to bite them in the ordinary way.

In other words when the fly is able to transmit Sleeping Sickness by biting an animal, its alimentary and salivary contents are also injective when infected with a hypodermic syringe.

IV.—THE RESERVOIR OF THE VIRUS OF SLEEPING SICKNESS.

Reference has already been made to the fact that it was believed that the way to stamp out the infectivity of the epidemic areas was to remove all the human population from the fly zone on the islands in and on the shores of the great Lake. Acting on this advice the Administration had done this, and when we began to work on the subject the lake shore had been deserted for about two years and most of the islands had been cleared of human population. No one was allowed within two miles of the water edge or on its water under pain of penalty. One of our first experiments was designed to ascertain how far these measures had succeeded in sterilizing the fly population and we certainly were not prepared for the result.

We found that when batches of tsetse flies were caught at various parts of the deserted lake shore and were brought up and fed on healthy monkeys that these monkeys invariably became infected with Sleeping Sickness.

The surprising conclusion was " that the *Glossina Palpalis* on the uninhabited shores of Victoria Nyanza can retain their infectivity for a period of at least two years after the native population has been removed."

There were four ways in which this might be accounted for :—

Firstly, the flies may occasionally get access to stray natives passing from the islands to the main land or others who have evaded the regulations.

Secondly, our own fly boys and canoe boys who go to catch the flies might have been suffering themselves from Sleeping Sickness.

Thirdly, the life of the *Glossina Palpalis* may be longer than two years and the flies which were now proved to be infective may have become so two years or more ago.

Fourthly, it is possible that some animals or birds on the lake shore are infected and so constitute a " reservoir " of the disease.

We were able to satisfactorily exclude the first two possibilities ; we could not deny the third though it seemed improbable.

We thought that the reservoir theory was most likely to be the explanation and work was commenced in a variety of ways to prove this. As a result of these experiments it is not too much to say that a new chapter has been opened in the epidemiology of Sleeping Sickness as the succeeding pages will show.

The proof of the existence of a " reservoir " is not scientifically complete but there is little doubt of its accuracy.

The important part which is played by wild animals in keeping up local infectivity to trypanosome disease was discovered by Bruce in Zululand and will be found detailed in his classical researches into Nagana or the tsetse fly cattle disease of South Africa.

He found that the infectivity of the tsetse fly (*Glossina Morsitans*) was kept going by wild animals, particularly buffaloes, which frequented the cattle grazing grounds and which harboured in their blood the *Trypanosoma Brucei* and which every now and again broke out in virulent epidemic form amongst the domestic stock.

The phenomenon of " reservoirs " is comparable with that of carriers of enteric, diphtheria and other diseases, a problem which now-a-days is so prominently occupying the attention of epidemiologists in many parts of the world.

It amounts to this that certain individuals who themselves show no symptom of disease are carrying about a supply of virus wherewith epidemics may be lit up in appropriate surroundings.

In trypanosome reservoirs the animals which carry the virus are probably racially immune, whereas as in the above disease of man the immunity is only a personal one and acquired as a result of an attack of the disease from which the individual has recovered.

To return to the subject of Sleeping Sickness the question was—Does some reptile, bird or mammal living on the lake shore act as a reservoir of the virus of Sleeping Sickness ?

An answer to the question was sought in various ways :

(*a*) The blood of various creatures could be searched in the hope of finding *Trypanosoma Gambiense.*

(*b*) The blood of such creatures could be injected at once after death into susceptible animals.

(*c*) Blood containing *Trypanosoma Gambiense* could be injected into various animals and the fate of the trypanosomes studied.

(*d*) Animals considered likely to be " reservoirs " might be artificially infected with Sleeping Sickness and an attempt made to transmit the disease from them to healthy animals either by inoculation of blood or better still by using laboratory bred tsetse fly to carry the infection from the sick to the healthy animals.

All these methods were used.

For the first year or so of the time in Uganda all manner of creatures, lizards, snakes, amphibians, birds, and mammals were killed and their blood subjected to careful scrutiny.

The result was that a regular museum of blood parasites was obtained, but neither the *Trypanosoma Gambiense* or any very near ally was found. A number of blood films were sent to England and were entrusted to the care of E. A. Minchin, M.A., Professor of Protozoology in the University of London, who kindly described them.

His paper (No. 6), which is beautifully illustrated from his own and Lady Bruce's drawings, may be studied by those interested in blood parasites, but as it has no direct bearing on the subject of this paper nothing further need be said about it.

A paper (No. 1, *vide* Summary, page 23) entitled " *Trypanosoma Ingens* " contains an interesting observation on the subject of " reservoirs." This huge (amphibian like) trypanosome was found in the blood of cattle and also in bush buck, a species of wild antelopes, which frequented the same feeding ground as the cattle.

It is quite possible that one of these animals was acting as a reservoir to the other, but as neither seemed to suffer from the effect of the trypanosome, the subject in this particular instance is of academic interest rather than of practical importance.

The second method of injecting the blood of a newly killed animal into a susceptible animal was also carried out in a number of instances, but without success. In most cases it was the blood of lake shore birds that was used, but that of hippopotamus, crocodile and antelope was also injected. It was not surprising that this method failed because where reservoirs exist they generally only form a very small proportion of the class of animals they represent. Then the blood has to be injected at once into the test animal, which in this case meant that monkeys had to be carried about on all shooting expeditions. This involved the fallacy that by taking a susceptible animal into a fly belt known to be infective it is liable to contract the disease naturally.

The animals to which most attention was paid were those found most frequently on or near the lake shore. As a representative of the birds the common fowl had to be used, as it was not found possible to experiment with the divers, cormorants and other species actually found on the lake shore.

Next the domestic cattle were examined.

Lastly the wild antelope which infest the shores were subjected to special attention.

A number of attempts to transmit Sleeping Sickness by means of laboratory bred tsetse flies from infected or potentially infected fowls to clean monkeys were made—but with negative results.

The conclusion was that the domestic fowl cannot act as a reservoir of the virus of Sleeping Sickness, but we pointed out that this negative result does not necessarily apply to the birds found naturally in the fly area.

Native cattle were then examined. The importance of the animals being a reservoir is obvious, for they go down to the water's edge daily to drink and generally live amongst the people in or about the kraals. They might act readily as a go-between if proved to be susceptible to infection with *Trypanosoma Gambiense.*

The experiments detailed in Paper No. 8 (*vide* Summary, page 23) prove that cattle are susceptible to *Trypanosoma Gambiense.* The facts brought out may conveniently be presented in the form of question and answer :—

Question 1.—Are cattle capable of being infected with Sleeping Sickness by the subcutaneous injection of blood containing *Trypanosoma Gambiense ?*

Answer.—Yes ; they may be infected in this way. The trypanosome appears in small quantities in the blood, and the blood when injected into susceptible animals, such as monkeys, gives rise to a fatal form of the disease in them.

Question 2.—Can cattle be infected with Sleeping Sickness by the bites of artificially infected *Glossina Palpalis ?*

Here the tsetse flies were first fed on infected monkeys and after the necessary interval on an ox day by day.

Answer.—Two experiments were performed to show that cattle could be infected in this way and that their blood was infective to monkeys.

Question 3.—Can cattle be infected with Sleeping Sickness by the bites of naturally infected flies caught on the lake shore ?

Answer.—Three experiments proved this possible and again the blood of the cattle was proved to be infective to monkeys and to a goat when it was injected into them.

Question 4.—Is it possible to infect tsetse flies by feeding them on cattle suffering from Sleeping Sickness and afterwards to transmit the disease by means of these flies to healthy animals ?

This experiment approximated still more to natural conditions of infection and nothing but laboratory bred and therefore " clean " flies were used.

Five experiments were done ; three were negative and two were positive.

The experiments are so important that one will be given in detail to show how it was done.

Experiment 1602.—Fifty laboratory bred flies were fed for four successive days on an ox whose blood contained *Trypanosoma Gambiense.*

After a period of starvation they were applied to a monkey and a goat on alternate days.

The monkey died (accidentally) before it could have become infected, but the goat developed Sleeping Sickness 20 days after the flies had had their first infected feed.

The remainder of the flies, 32 in all, were dissected and five were found to contain flagellates. The alimentary contents of one of these flies were injected into a monkey and after an incubation period of 13 days *Trypanosoma Gambiense* appeared in its blood.

Answer.—Laboratory bred flies can be infected by feeding them on cattle infected with Sleeping Sickness and afterwards the disease can be transmitted to healthy animals by means of these flies.

Question 5.—Do cattle when living in the fly areas actually carry the virus of Sleeping Sickness ?

This is the last link in the chain of evidence and again it was positive.

One out of 17 cattle which were presumed to have been exposed to the bites of *Glossina Palpalis* was found to contain *Trypanosoma Gambiense* in its blood.

Answer.—Yes ; cattle in their natural state and apparently in good health may harbour the virus of Sleeping Sickness.

As a final conclusion to this very important paper it was stated that it is possible that cattle and antelope living in the fly area may act as " reservoirs " of the virus of Sleeping Sickness and so keep up the infectivity of the tsetse fly.

The next Paper (No. 18, *vide* Summary, page 23) shows how the problem was applied to the antelope.

There are many antelope living on the lake shore besides buffaloes, hippopotami and other game, and now that the human population has been compulsorily removed the wild animals are greatly increasing in number, so much so that the two-mile zone is becoming a huge game reserve. The experiments with antelope were carried out on partially tamed animals of various species caught when young.

A freshly recovered strain of human trypanosome was used and only laboratory bred flies were used.

The steps of the scheme were very similar to those described in the last paper and the following questions were set for solution :—

I. Can antelope be infected with Sleeping Sickness by the bites of laboratory bred and laboratory infected *Glossina Palpalis ?*

Eleven experiments were done and all were positive ; the answer is therefore in the affirmative.

II. If antelope can be infected with the virus of Sleeping Sickness, can they transmit the infection to clean laboratory bred *Glossina Palpalis* when these tsetse flies are allowed to feed on them ?

Further if these *Glossina Palpalis* become infected, can they transmit the virus to susceptible animals ?

To answer the question 24 experiments were done, and of these 17 were positive (70·84 per cent). The shortest time before the flies became infective was 24 days and the longest 49 days. It is interesting to note that tsetse flies were more readily infected from antelope and in a larger number than any other kind of animal used by the Commission.

The observers add :—

" The most significant of the above observations is the one in which it is shown that 55 days after the last feed of infected *Glossina Palpalis* on bush buck (Experiment 2328) the blood of this buck was capable of infecting clean laboratory bred flies though *Trypanosoma Gambiense* had never been seen in its blood.

III. If *Glossina Palpalis* can be infected with the virus of Sleeping Sickness by feeding on the blood of *Trypanosoma Gambiense* infected antelope, what percentage of flies are found to be infected?

In the sixteen positive experiments 1,102 flies were dissected, and of these 119 (54 ♂ 65 ♀) were found to be swarming with developmental forms of *Trypanosoma Gambiense*, that is about 10 per cent.

IV. How does Sleeping Sickness affect the health of the antelope?

They do not seem to suffer any inconvenience from the presence of the trypanosome.

The final question was—Are antelope living in the fly area naturally infected with Sleeping Sickness?

A large antelope drive had been arranged to try and prove the point, but the Commission left before it was carried out.

The conclusions which terminate this important paper are given as below:—

1. Water buck, bush buck and reed buck can readily be infected with a *human strain* of the trypanosome of Sleeping Sickness by the bites of infected *Glossina Palpalis*.
2. One exposure to the bites of infected flies is sufficient to infect an antelope with the virus of Sleeping Sickness.
3. Though the blood of an antelope may be proved to be infected with *Tr panosoma Gambiense*, careful and continued examinations over prolonged periods may fail to reveal the presence of the parasite in the blood.
4. The incubation of the disease in the antelope is probably seven days.
5. Antelope of the water buck, bush buck and reed buck species, when infected with the virus of Sleeping Sickness, can transfer the infection to clean laboratory bred *Glossina Palpalis*.
6. This transmission of the infection to clean laboratory bred flies may occur at least 81 days after the last feed of the infected flies on a buck.
7. *Glossina Palpalis* when infected with the virus of Sleeping Sickness obtained from the blood of infected antelope, are capable of transmitting the virus to susceptible animals.
8. An appreciable percentage of *Glossina Palpalis* will become infected with the virus of Sleeping Sickness should these flies feed on antelope suffering from this disease.
9. It follows from the above conclusion that antelope living on the fly areas are "potential" reservoirs of the virus of Sleeping Sickness.
10. No antelope up to the present has been found naturally infected with *Trypanosoma Gambiense*.

Although the last link in the chain of evidence is missing, few people who read the above evidence or, better still, the original paper, will have much doubt as to the evil potentialities of the antelope as a reservoir of Sleeping Sickness.

Treatment.

It is outside the scope of this paper to say anything on the result of treatment especially as with one exception the Commission did not touch on the subject.

Suffice it to say that though many drugs have the power of driving trypanosomes out of the blood stream for the time being, none can be credited with

effecting a permanent cure except perhaps in the very early stage. The course of the disease in man may be very prolonged and persons apparently cured have succumbed to the disease even eight or ten years after infection. Sleeping Sickness is still a disease with a mortality not far off an hundred per cent.* One series of experiments was done by the Commission to ascertain whether treatment of Sleeping Sickness patients with the usual trypanocidal drugs rendered them non-infective to tsetse flies, and as the question involved is one of great practical importance the experiment will be now outlined.

In this case the *Trypanosoma Gambiense* was obtained directly from man instead of from animals infected in the laboratory. Firstly, an attempt was made to transmit the disease mechanically by the method of interrupted feeding from infected men to monkeys.

Those who read the previous remarks on the subject of mechanical transmission will not be surprised to find that all the fifteen experiments were negative. Secondly, an attempt was made to transmit the disease from Sleeping Sickness patient to monkey after the parasite had completed its development in the fly.

Six experiments were carried out on patients in whose glands trypanosomes had been found at some time or another but who still appeared to be in good health.

All had received trypanocidal treatment. None of the tsetse flies became infected, that is to say, all the experiments were negative.

In the next series patients were used who were in a more advanced stage and in whom the disease might reasonably have been diagnosed on clinical grounds alone.

Twenty-five patients were the subject of the observation and of these six were untreated and the remainder had had trypanocidal drugs.

All the treated and four of the untreated proved non-infective, that is, only two patients, and those both untreated, proved to be infective to the flies which in their turn passed on the infection to clean monkeys. Finally, some experiments were done on patients on an advanced stage of Sleeping Sickness. Ten experiments were done and one was positive. This patient had been under treatment for a month and had been given during that time two grammes of soamin and 0·08 gramme of Perchloride of Mercury.

Seventy-four flies from this positive experiment were dissected and four flies were found to contain flagellates on the 28th, 39th, 40th and 46th day, respectively, after their first infected feed.

We may therefore conclude that—

Firstly, Glossina Palpalis fed on natives suffering from Sleeping Sickness and untreated by drugs may become infected and be capable of transferring the disease to healthy animals.

Secondly, that *Glossina Palpalis* fed on natives suffering from Sleeping Sickness and treated by arsenic and other drugs may also become infected and capable of transmitting the disease to healthy animals.

The prevention of Sleeping Sickness.

This may be attempted by attacking any one of the three factors on which the disease depends—

(*a*) The human factor.

(*b*) The tsetse fly.

(*c*) The reservoir of infection.

*These remarks apply particularly to the disease as is met with in Uganda and surrounding countries. It seems that the West African type is not so dreadfully fatal.

In the past the pandemic of Sleeping Sickness in Africa has been caused by the opening up of trade routes and by human intercourse, and it is by legislation applied to human beings that the principal efforts are being made to check its progress.

The infected areas are pretty accurately known and to a less extent the areas of potential epidemicity, the latter, in our present state of knowledge, being taken to coincide with the distribution of *Glossina Palpalis* or at any rate of tsetse flies in general. The enlargement of the cervical lymphatic glands is taken as a working diagnostic sign of the disease in the earlier stage and all persons possessing this sign are forbidden to enter non-infected areas.

This is an excellent prophylactic method as far as it can be carried out, but the loopholes are numerous.

Clearing the epidemic areas of human inhabitants is effective up to a point, but it means handing over the position to the enemy, and the present condition of the lake shore as our experiments show is probably worse than the first.

It also has the economical disadvantage of giving back some of the most fertile parts of the country to the jungle.

The prophylactic administration of drugs to the native has not been tried on a large scale, but from our experiments just referred to it would probably be ineffectual.

Man does not appear to be capable of responding to the virus of Sleeping Sickness by producing any marked active immunity, so that channel of prevention does not seem hopeful.

It is probable that the West Africa natives may have in generations acquired some immunity, but there is no way yet known in which this can be hastened.

Is it possible to stamp out Sleeping Sickness by exterminating the tsetse fly?

Without answering that question one may ask another. Has anyone succeeded in stamping out plague by exterminating the rat or the flea? It is true that the tsetse fly may be got rid of in small areas by laying the country bare of trees and all jungle, but this as a general measure applicable to wide areas of infection is quite impracticable and may be dismissed as such.

The third method of prophylaxis is that directed towards the destruction of the "Reservoir." If the antelope and other wild game in the fly zone are carrying and disseminating the virus of Sleeping Sickness, it is evident that they will have to be exterminated.

It seems unfortunately true that the presence of game in any number is uncompatible with the march of civilization, and this applies as much to stock raising in general as it does to Sleeping Sickness in man.

The new Commission, which has just gone to Nyassaland to investigate the question of Sleeping Sickness there, will make this question of animal reservoirs their special study.

CAN SLEEPING SICKNESS GET A FOOTHOLD IN INDIA?

This extremely important question is one to which it is not possible to give a decided answer: the probabilities are against it, but it would be unwise to neglect precautionary measures. In another part of the paper it was stated that Sleeping Sickness had not been known to spread in the proved absence of tsetse flies, and authorities like Koch and Bruce have given decided opinions against the possibility of the disease spreading by the agency of any other insect. Mr. H. Maxwell Lefroy, Entomologist to the Government of India, states in the "Preliminary Account of the biting flies of India," p. 41 :—

".....There is no record yet of *Glossina*, the tsetse fly of Africa, nor is there at present any reason to believe that it occurs in India; in fact the work of

the last two years goes to confirm the belief that this genus will not be found in India."

If this is true and if it is also true that the tsetse fly is a *sine quâ non* in the distribution of the disease then India is safe. Considerable support is given to this belief by the results of a natural experiment done on a large scale in the early part of the last century. In the days of the slave trade between the West Coast of Africa and the West Indies and the Southern States of North America negroes stricken with Sleeping Sickness were imported into the New World. The disease however did not spread, for although one may assume the presence of many parasitic insects the tsetse fly did not exist there. At the same time against this optimistic outlook one must remember that tsetse flies can live in other parts of the world besides Africa. They have been found in Arabia and some pupæ which we sent home from Uganda to the Zoological Gardens, London, hatched out as healthy insects.

There are many Indians living in Uganda and doubtless in other endemic centres of infection and most of these return sooner or later to settle in their native land.

It is highly probable that from time to time the disease will be in this way imported into India from East Africa or the Soudan to the ports on the west side of India.

Importation by Sea.

The Government of India are fully alive to this possibility and *Regulations to prevent the importation of Sleeping Sickness into India* were made as far back as 1903. In 1908 fuller precautionary measures were instituted and these are at present in force pending new rules relating to Sleeping Sickness which are to be promulgated as the revised uniform rules under the Indian Ports Act, 1908, for all infective and contagious diseases. The rules now in force are briefly as follows.

If a case or suspected case of Sleeping Sickness or a death from the disease has occurred on board any vessel coming from a port beyond India, the master of the vessel shall report the same to the Boarding Port Officer. The Health Officer shall be immediately informed and he shall proceed on board and carry out the necessary examination. If any person is suspected to be suffering from Sleeping Sickness he shall be removed to a hospital and "shall be isolated in such a manner as will prevent, as far as possible, the communication of the disease to persons by the agency of biting flies and shall continue to be so detained and isolated until a Medical Officer authorised by the local Government on this behalf certifies that he is not suffering from Sleeping Sickness, or that having suffered from the disease he has been cured." In the revised Regulations referred to which are under consideration the rules are as follows:—

33. In the case of a vessel having on board a person suffering, or suspected to be suffering, from Sleeping Sickness, the person or persons shall not be permitted to land without the specific written permission of the Health Officer, who may, pending the receipt of written instructions from the local Government, permit the landing of such persons only if arrangements can be made for their strict isolation on shore.

Note.—*In suspicious cases* the Health Officer should make a careful examination for enlargement of the lymphatic glands, particularly those in the triangles of the neck. The pulse rate should also be taken. To confirm the diagnosis gland-juice should be taken and examined for the presence of typanosomes.

34. In the case of a vessel arriving from the East Coast of Africa within the limits of Port Sudan, Durban, or from other localities declared to be infected, the procedure prescribed by rule 2 shall be complied with and the crew or passengers, etc., shall be medically inspected in accordance with rule 9.

These regulations may be considered adequate as far as they go and should certainly not be relaxed in any particular. If a quarantine station is needed the suitability of one of the islands in Bombay harbour (*e.g.*, Butcher Island) should be considered, as it is improbable that much interchange of biting insects takes place between these islands and the main land.

All the Soudan and Somaliland ports should be looked upon as possible sources of infection as well as those of the East Coast of Africa.

The movements of cattle and camels and other animals between Africa and India should be watched in view of the fact that some may be acting as carriers of the infection of Sleeping Sickness.

The Pilgrim Traffic Question.

The question may arise at some time as to whether similar precautions should be taken with reference to the return of pilgrims from Arabia to India. Large caravans of Muhammadans gravitate to Mecca from the West Coast of Africa and the endemic centres of Sleeping Sickness, and as a tsetse fly is known to exist in Arabia there are the necessary conditions for the dissemination of the disease.

If this did occur it would mean that pilgrims in the early and most infectious stages of the disease might be distributed all over India. However, so far, Sleeping Sickness itself is not known to have occurred in Arabia, so that it may not be necessary to do more at the present time than to keep in mind the possibility of its so doing.

ADDENDUM.

Since the main body of this paper was written a great deal of work has been done both in the infected areas and in European laboratories to clear up the questions concerning the identity of the new Rhodesian trypanosome and its carrier.

Firstly, it has been shown that *Trypanosoma Rhodesiense* (Stephens and Fantham) is not identical with *Trypanosoma Gambiense*. This conclusion was arrived at by the consideration of certain morphological differences; by the greater virulence of the former trypanosome for laboratory animals than the latter possesses and also by means of the "crossed immunity test." The last named test showed that an animal which was immune to T. *Gambiense* was still susceptible to and died as the result of an inoculation with *T. Rhodesiense*.

Secondly, it was urged that *T. Rhodesiense* was in reality the well known cattle trypanosome of South Africa (*T. Brucei*) which is naturally carried by *Glossina Morsitans* and that this parasite had recently become pathogenic to man. Laveran from evidence obtained by his crossed immunity test and by serum reactions came to the conclusion that the two were distinct species. The evidence of morphology and of animal reaction also goes to disprove the identity of these two trypanosomes.

Thirdly, can *G. Morsitans* (the cattle tsetse fly) transmit the Uganda form of Sleeping Sickness? It has been proved by many laboratory experiments that *T. Gambiense* can be readily conveyed to monkeys and other animals by *Glossina Morsitans* and that it was, therefore, possible that the fly could take the place of G. *Palpalis* in areas unsuited to the life of that insect.

At the same time opinions have been expressed that other tsetse flies, *Glossina Fusca* and *G. Brevipalpis* may be found to be capable of transmitting human trypanosomiases either in Rhodesia or elsewhere. There is no evidence to show that in nature the *virus* of Uganda Sleeping Sickness (*T. Gambiense*) is carried by any tsetse fly except *G. Palpalis* though laboratory experiments show that in theory other tsetse flies can do so.

Finally, there remains the question of how the Rhodesian trypanosome infects man. We have seen that this trypanosome is distinct both from *T. Gambiense* and *T. Brucei*, and we know that it is transmitted in areas free from *Glossina Palpalis* but which are infested with the other common tsetse fly *Glossina Morsitans*.

Stephens and Fantham sum up their observations (January 7th, 1912) as follows:—"It appears to us that the foregoing evidence is strongly confirmatory of the view we put forward in our original paper, namely, that we have two trypanosomes producing Sleeping Sickness in man, namely, *T. Gambiense* and *T. Rhodesiense* and that the carrier of the latter species is *Glossina Morsitans*."

In the Sleeping Sickness Bulletin which has just come to hand (No. 37, Volume 4, published May 11th, 1912) the report of the Luangwa (Rhodesia) Sleeping Sickness Commission is reviewed and the conclusions arrived at by Drs. Allan Kinghorn and Warrington Yorke are so important that they will be quoted in full.

"1. The human trypanosome, in the Luangwa Valley, is transmitted by *Glossina Morsitans*, Westw.

"2. Approximately 5 per cent. (4·76) of the flies may become permanently infected, and capable of transmitting the virus.

"3. The period which elapses between the infecting feed of the flies and the date on which they become infective is approximately fourteen days.

"4. An infected fly retains the power of transmitting the disease during its life, and is infective at each meal.

"5. Mechanical transmission does not occur if a period of twenty-four hours has elapsed since the infecting meal.

"6. Some evidence exists to show that in the interval between the infecting feed and the date on which transmission becomes possible the parasites found in the flies are non-infective.

"7. *Glossina Morsitans*, in nature, has been found to transmit the human trypanosome.

"8. Certain species of buck, *viz.*, waterbuck, hartebeest, mpala, and warthog, have been found to be infected with the human trypanosome.

"9. A native dog has been found to be infected with the human trypanosome."

It is interesting to note how closely the results obtained by these observers with the Rhodesian trypanosome tally with ours and the Uganda strain for, substituting *Glossina Morsitans* for *Glossina Palpalis*, the other facts are almost identical with those which we elicited.

To summarise the whole position in a few words.

There are two distinct human trypanosome diseases found in adjacent parts of Central Africa ; one the Uganda form carried by *Glossina Palpalis*, the other the Rhodesian form carried by *Glossina Morsitans*.

Both diseases are spreading outside their original epidemic area and they may continue to spread in those countries wherever the appropriate fly exists.

In both cases a reservoir of infection exists in the wild game, and the tsetse fly carries the disease from game to man, and from man to game (possibly also from man to man) and in this way keeps up a vicious circle.

If these new facts have any further bearing in India other than that already discussed, they should urge us to strengthen rather than to weaken our defences against the possible inroads of this dreadful disease.

F. P. MACKIE.

SHILLONG :

The 26th June 1912.

A list of the papers dealing with the work of the Sleeping Sickness Commission of 1908—1910 published in the proceedings of the Royal Society during 1909, 1910 and 1911.

B. Volume 81.

1. *Trypanosoma Ingens, Nov. sp.*
2. The development of *Trypanosoma Gambiense* in *Glossina Palpalis.*
3. A note on the occurrence of a Trypanosome in the African Elephant.

Volume 82.

4. Sleeping Sickness in Uganda. Duration of the infectivity of the *Glossina Palpalis* after the removal of the Lake-shore population.
5. *Glossina Palpalis* as a carrier of *Trypanosoma Vivax* in Uganda.
6. Report on a collection of blood-parasites made by the Sleeping Sickness Commission, 1908-09, by E. A. Minchin, M.A., Professor of Protozoology in the University of London.
7. *Amakebe*, a disease of Calves in Uganda.
8. The Development of Trypanosomes in Tsetse flies.
9. Experiments to ascertain if cattle may act as a Reservoir of the Virus of Sleeping Sickness.
10. Trypanosome diseases of domestic animals in Uganda. I—*Trypanosoma Pecorum.*
11. The natural food of *Glossina Palpalis.*
12. "*Muhinya,*" a disease of natives in Uganda.
13. Mechanical transmission of Sleeping Sickness by the Tsetse fly.

Volume 83.

14. Trypanosome diseases of domestic animals in Uganda. II—*Trypanosoma Brucei* (Plimmer and Bradford).
15. Trypanosome diseases of domestic animals in Uganda. III—*Trypanosoma Vivax* (Ziemann).
16. Trypanosome diseases of domestic animals in Uganda. IV—*Trypanosoma uniforme* sp. nov.
17. Trypanosome diseases of domestic animals in Uganda. V—*Trypanosoma Nanum* (Laveran).
18.* Experiments to ascertain if the domestic fowl of Uganda may act as a Reservoir of the Virus of Sleeping Sickness (*Trypanosoma Gambiense*).
19.* Experiments to ascertain if Antelope may act as a Reservoir of the Virus of Sleeping Sickness (*Trypanosoma Gambiense*).
20. Experiments to ascertain if *Trypanosoma Gambiense* during its development within *Glossina Palpalis* is infective.
21. † Experiments to investigate the infectivity of *Glossina Palpalis* fed on Sleeping Sickness patients under treatment.
22. * Experiments to ascertain if certain *Tabanidæ* act as the carriers of *Trypanosoma Pecorum.*
23. Further researches on the development of *Trypanosoma Gambiense* in *Glossina Palpalis.*

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* Published under the names of the first three members of the Commission only.

† In association with Dr. R. Von Someran, Uganda Medical Staff.

PART VI.

CHOLERA.

ALL-INDIA SANITARY CONFERENCE—MADRAS—NOVEMBER 1912.

THE EPIDEMIOLOGY OF CHOLERA.

BY

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1. *Prevalence of cholera.*—Cholera has been known and dreaded as an acutely infectious and rapidly spreading epidemic disease since the beginning of recorded history, and even now it takes its place as one of the chief causes of death in the returns of vital occurrences in spite of our knowledge of its cause and in spite of the fact that the disease is admittedly preventible.

A reference to the mortality returns for the whole of India shows that during the 34 years from 1877 to 1910 there were nearly 13 million deaths reported from cholera, and it is interesting to note that while the first 17 years show a total of 6,052,179, the second period of 17 years shows 6,910,990 deaths, or nearly 900,000 more, and the last six years of all,—from 1905—1910, show a total of 2,816,376. These periods give annual averages of 356,000, 406,000 and 469,000 (omitting odd hundreds) respectively, and appear to show that cholera is becoming progressively more prevalent in spite of civilisation and sanitation.

I am aware that it may be argued that the apparent increase is due to more accurate reporting, but my experience in checking the actual returns of recent years has shown me that even now the returns are considerably below the actuals especially in times and places of acute epidemics. I am unable to speak from experience of the reliability of the earlier returns, but it seems to me probable that the variations are not likely to have been great, because cholera is and always has been a well known disease, with definite characteristics, and the error in reporting by an uneducated agent, such as the village *chowkidar* who has not changed much in his educational or social status in 34 years, is more likely than otherwise to be approximately a constant factor in dealing with large numbers.

The inference therefore is that cholera is really becoming progressively more prevalent, and the probable explanation is the improvement in the means of communication and transit, which has taken place during these 34 years, and which makes the distribution of the disease so much more rapid and more easy.

With regard to the localisation of the disease, it is to be noted that Bengal has always been the home of epidemic cholera and claims 5,426,417 out of the total number of 12,963,169 deaths reported during the period.

With regard to the distribution of the disease, it may be noted that it is universal all over India but by no means in proportion to the populations of the various provinces. The provinces which have suffered most from cholera are Bengal, the United Provinces and Madras and these provinces are characterised by two of the conditions to which I shall later refer as being intimately associated with the epidemic occurrence of cholera; *viz.*, crowding or a high rate of density of population, and a favourable climate, which is both warm and moist during a great part of the year.

Whilst discussing the prevalence of cholera we may also with profit compare its prevalence and its mortality with other great epidemic diseases, such as smallpox and plague, and incidentally we may note the differences in the preventibility of these diseases, and what has actually been done to diminish their prevalence. I do not propose to enter into a detailed and lengthy comparison but merely to compare the general epidemiological characteristics.

The point of importance seems to me to be that cholera occurs and recurs with undiminished virulence, and causes a large number of deaths every year, and

frequently causes a very large number. In the 34 years from the records of which I have been quoting, the number of reported deaths has been over half a million in eight separate years and over 400,000 in fourteen years. Now in comparison with small-pox this mortality is much greater, and in comparison with plague it is greater in the aggregate, though it has been surpassed in single years. The important points are the permanence of cholera as compared with plague and its continued and sustained virulence as compared with small-pox which is a decadent disease.

Now these three diseases are all more or less preventible, small-pox altogether so, cholera to a large extent, and plague to some extent, and they should merit the attention and efforts of the sanitarian in direct proportion to their preventibility, with some consideration to their prevalence and mortality.

In the order of known preventibility we have small-pox, cholera, plague. In the consideration of their prevalence and importance as causes of sickness and death, we find them in order: cholera, small-pox, plague.

A general consideration of the importance of applying preventive measures to these diseases on a large and general scale would lead one to deal with cholera and small-pox as the most important, and plague as a disease of secondary importance. Instead of this however being the case we find that small-pox receives regular and systematic attention, and plague is the absorbing subject to which excessive importance is attached, and upon which enormous effort is concentrated, whilst cholera is, like Cinderella, ignored and passed by.

This policy appears to me to be an erroneous one, the enormous economic importance of cholera and its persistent and increasing prevalence, combined with the admitted fact that it is largely preventible, demand that it should receive more serious attention and point to the necessity for a systematic policy and a definite organisation for its prevention.

2. *The History of Cholera Investigation.*—Little was really known about cholera until comparatively recent years, except that it was an acutely infectious disease with a very high death rate.

The previous generation of medical officers in India had a large practical experience of the disease, and elaborated an air borne theory which survived and held the field until the occurrence of the historic epidemic at Hamburg-Altona, which led to a revision of existing ideas and the adoption of a water borne theory on a hypothesis, which does not logically apply and by a process of argument which is the reverse of logical.

Let us first examine the air borne theory of the older school, and let us begin by admitting that the men who elaborated it were probably not fools, and that their opinions were founded on observed facts and merit consideration.

Some years ago, I read a paper on this subject by Surgeon-General Cunningham, in which he traced the history of several epidemics and showed that cases occurred in localised areas at first and spread principally in the definite direction of the prevailing wind, which is almost a constant factor in the United Provinces in summer. He further showed that neighbouring houses were usually the first to be infected though the infection frequently jumped over one or more houses, and he laid stress on the fact that the water-supplies were wells which were entirely separate and very unlikely to become contaminated because no one living outside the premises had access to them. He therefore argued that the infection was not water borne but must be air borne and he deduced that the easiest way to avoid the infection was to go away and live somewhere else to windward of the epidemic. The argument is apparently a logical one and was justified by the results of the procedure which he recommended for his troops when epidemics occurred. The only factor which Surgeon-General Cunningham omitted from his argument was the fly, and with the assistance of that prevalent and audacious insect, I propose to revive the theory and elaborate the argument,—with modifications.

Before leaving the theories of infection and their history I propose first of all to discuss the Hamburg-Altona epidemic, and its influence on the minds and practice of almost a generation of medical men. The circumstances are fairly well known but may be summarily repeated.

Hamburg and Altona are adjoining towns on the banks of the river Elbe, and both took their water-supply from the river, but the Altona supply was well filtered before being distributed, whilst the Hamburg supply was not filtered.

A fulminating epidemic suddenly broke out in Hamburg but Altona remained largely free from the disease. It may be added that cholera was at the time epidemic in Russia and Eastern Germany. The obvious explanation was that the river water had got contaminated, and as the Hamburg supply was taken at a point higher up than the Altona supply it was also obvious that the filteration was the thing that saved Altona.

The matter was clinched by the isolation of the cholera vibrio from the Hamburg water, and river water, and by the fact that it did not occur in the Altona filtered water. So far, so good.

The fact was proved that cholera infection could be conveyed in water; but the fallacy was accepted that cholera is a water borne disease, which is quite a different proposition.

I maintain that nothing more was actually proved than the possibility of cholera being water borne: nothing more could be proved from any such single example: and I maintain that the water borne theory is based on the obsession of an illogical and erroneous idea which has been accepted and raised to the dignity of a theory of transmission because of the clearness and completeness of proof in an isolated case and because of the great publicity which it received.

3. *The probable source of Cholera Infection.*—That there is a perennial and persistent source of cholera infection, that survives throughout the centuries under many various circumstances, and at all times, is indisputable, and, in order to arrive at a logical hypothesis with regard to the transmission of the disease, it is obviously necessary to begin with a definite and intelligible theory of its source.

I do not wish to trespass too far into the domains of the bacteriologist, but I must do so to some extent in order to establish and elucidate my argument.

The known facts about the life history of the vibrio are many, but those which are important to my purpose are, that the organism is a delicate one and rapidly dies out when exposed to the sun-light or subjected to dessication, and that it is not capable of maintaining the struggle for existence for any long period against the more robust saprophytic organisms which swarm in all ordinary water-supplies.

It would therefore appear probable that the disease should be a rare one, and should tend to die out altogether. But such is not the case, therefore the logical explanation of its persistence must be sought elsewhere. Such an explanation is supplied by the recent work on typhoid fever which has given us the "Human Carrier Theory" to account for the persistence and recrudescence of the disease, and by the observations and experiments of a Russian bacteriologist who isolated the organism from the stools of healthy pilgrims who had returned from Mecca.

In other words, the obvious and satisfactory hypothesis is that cholera is also disseminated by the "Human Carrier", who may or may not have had the disease in a definite form, but who has the organism present and persistent in his intestinal tract for months and perhaps for years.

This theory does not clash with the known facts as to the feeble vitality of the organism outside the human body, but presents a complete and satisfactory explanation of all the known facts as to the outbreak and recurrence of the disease, and is intelligible and reasonable by analogy in comparison with

the proved theory of "Human Carriers" in enteric fever, which is in some respects a similar disease, and whose organism is also a delicate one, unable to live long under natural conditions outside the human host.

4. *The Factors of Transmission of Infection.*—If we accept the theory of carriers as the chief source of infection, it is easy to understand the occurrence of sporadic cases of the disease as well as the outbreak of great epidemics. The sporadic case is a constantly and vicariously recurring factor and epidemics occur when the conditions are favourable to transmission, and are therefore generally seasonal in their periodicity.

The means of transmission are probably several, but ultimately resolve themselves into the introduction of infected material into the stomach. This may be done either in food or in water, but probably occurs commonly in food, both because food is more liable to being handled by others than is water, especially in this country, and because in many forms it presents a suitable medium for the multiplication of the vibrio, which ensures that the organism is present in sufficient numbers before being swallowed, to produce an infection in some cases in spite of the bactericidal powers of the stomach.

The manner in which food or water becomes contaminated is comparatively easy to understand, when one considers the careless way in which the same water-supply is used for all purposes, and the negligent manner in which food stuffs are exposed to the contact of flies, and handled by servants and shop-keepers, whose personal cleanliness is a negligeable quantity, especially in the bacteriological sense.

5. *The Conditions Favouring Transmission.*—The conditions which favour transmission, and which are required to exist in a complete chain of circumstances if a great epidemic is to occur, are those which facilitate the rapid and frequent infection of food or water, and which provide a sufficient number of human hosts to receive the infection.

They may be summarised in an alliterative series as Carriers, Climate, Crowds, Contact, or Contamination of food or water, and bad Conservancy.

(i) *Carriers.*—The necessity for carriers as an original source of infection has been already discussed and it need only be added that the greater be the density of population in any area, the greater will be the number of carriers probably present, and the greater will be the probability of an outbreak if the other conditions are favourable.

(ii) *Climate.*—The condition of favourable climate is required for the outbreak of an epidemic in two ways,—partly because a rigorous climate tends to kill off the organisms in the excreta too rapidly to allow of frequent infection, but chiefly because a warm and moist climate is the one most suited to the rapid multiplication of flies, which I propose to credit with the chief part in the process of active dissemination of the infection.

(iii) *Crowding.*—Crowds or a condition of crowding or denseness of population are important chiefly from a mathematic point of view. In the first place the larger numbers present the possibility of more numerous sources of infection, and in the second place, the population is composed of units which act and react upon each other, every unit being a potential source of infection to every other unit, a condition which obviously makes the probability of infection proportionate to the square of the population in a crowded area, or rather, to be exact, equal to $N \frac{2}{=} N$, where N is the total number of the population.

There is also a further factor introduced by crowding, which is more difficult to measure, and which consists in the increased nearness produced amongst the units of the crowd, and the consequent increased chance of contact contamination of hands, clothing, or vessels.

6. *Contact or Contamination.*—The importance of contact is probably small as compared with contamination of food or water by other means, and only assumes dimensions deserving consideration when the crowding is excessive, as happens at some of the great *melas* in India.

The usual means of transmission of the infection are almost certainly through food and drink and the question of interest is,—how does the food or drink become contaminated ?

As far as water-supplies are concerned, they may be contaminated in this country with great ease owing to the rarity of filtered and piped supplies; the ordinary use of surface and river waters, and the extraordinary habits of the people who will wash, bathe and drink indiscriminately in the same water.

The logical conclusion would therefore appear to be that the entire population should die of cholera, and as this has not occurred at any time known or recorded, it may reasonably be presumed that the contamination of water is not the usual nor the most important means of infection.

Concerning the contamination of food which I consider to be the common cause of infection, I wish first of all to emphasise its suitability, in many of its forms for the purpose, inasmuch as it provides a medium upon which the implanted organism can live and multiply, until it is present in sufficient numbers to be dangerous Secondly it should be noted that food is usually left lying about uncovered because the ordinary inhabitant of India does not know any better, and in its prepared forms it is exposed in filthy bazars in the most careless manner. Further it is subject to more frequent handling than is water. It is therefore in every way a more suitable means of transmission and requires only that the infecting agent should be identified in order to prove its probability. That infecting agent is in most cases the fly, whose presence on the scene is due to bad conservancy, and whose history, habits, and importance in this connection will be discussed in a succeeding paragraph.

7. *Conservancy.*—The importance of conservancy in the question is an indirect one, but is nevertheless very real and very great.

The ordinary native of India is possessed of peculiarly promiscuous and primitive habits; and wherever he goes his habits go with him. Conservancy has to deal with and dispose of his excreta, and the goodness of the conservancy is in proportion to the promptness and completeness with which this work is accomplished.

Bad conservancy leaves this work undone—and this is where the fly comes on the scene.

Flies haunt the habitation of man because they most readily find the two essentials of life—a supply of food, and facilities for the reproduction of their species. What is food for man is generally, if not always, food for flies, and flies always try to take their share of it: further also they feed on substances of a filthy or putrid nature, and they utilise garbage and excreta, and by preference the latter, as a breeding ground in which to lay their eggs.

Bad conservancy therefore leads to the enormous multiplication of flies, and also offers increased facilities for contamination of the feet and intestinal contents of these flies in proportion to the degree of inefficiency of the conservancy.

8. *The Life History of the Fly.*—In order to make my argument more definite and more complete, I propose to submit a short resumé of some of the known facts in the anatomy and life history of the common fly, chiefly extracted from a paper by Dr. Graham Smith.

There are several species of fly but their anatomies and life histories are similar, and so they may be all taken together. The adult fly has a hollow proboscis with an oval suctional tip, and this communicates directly with a large crop or stomach. The fly is incapable of swallowing solid food, and when it feeds on any substance which is not sufficiently fluid to be drawn directly into the stomach, it repeatedly regurgitates and reabsorbs the fluid in its stomach, and thus slowly breaks up and absorbs solid matter either in solution or in suspension. This process largely explains the occurrence of contamination of food supplies. There is also however another additional explanation, which is that the fly is very greedy and always overloads its stomach whenever opportunity

occurs, and it subsequently vomits with considerable frequency in order to relieve the pressure. When one considers the habits of the ordinary fly—how it flies from garbage or excreta to food and back again—how it is exceedingly active and persistent in its progress—and how large and catholic is its appetite, these facts fully explain the great risk and probability of its carrying the infection of cholera, and demonstrate the importance of its function as a transmitting agent in cholera and other allied diseases.

The life history of the fly with special reference to the process and rapidity of reproduction are also very important. The female fly lays its eggs—generally over 100 in number—in garbage or dung, preferably in the latter: these eggs hatch out, and become mature most quickly under favourable conditions as to temperature and moisture, and subsequently develop more rapidly and vigourously when the food supply is plentiful. The eggs will not hatch at all below a certain temperature and hatch in from 8 to 24 hours according to the temperature. The egg hatches into a maggot which is very active and voracious, and feeds on the organic matter in which the eggs were laid. The maggot has a life of from 5 to 20 days according to the favourableness of the conditions, and then becomes a pupa for 5 to 14 days. From the pupa the adult fly emerges and is at once active, voracious, and sexually mature ready to give rise to another generation. The minimum period of development under favourable conditions is about 11 days and obviously the rate of multiplication of flies, and the occurrence of enormous numbers, will depend upon the existence of favourable conditions and food, and will be roughly in proportion to the "R" power of 50 where "R" represents the relative rate of rapidity of reproduction, and has a value ranging from unity—for the maximum period—to nearly three, for the minimum period of reproduction.

These facts explain the numerical prevalence of flies under various conditions, and demonstrate the importance of removing the facilities for their reproduction and nourishment during the process of development.

To complete the evidence against the fly it is only necessary to establish the fact that it can harbour living pathogenic organisms for a definite period. This has been done experimentally. Flies were fed on cultures of typhoid and tubercle bacilli and then removed from the source of infection and killed and examined in a daily series. It was definitely proved by repeated observations that the legs and wings were contaminated for a period of 12—24 hours, and that the stomach and intestines were contaminated for a period of 7 to 14 days, the living organisms were isolated, cultured and demonstrated from the crop and intestinal contents.

9. *The Importance of Separate Factors.*—When the chain of chance is complete, and the required conditions are all presented, a cholera epidemic may be confidently expected, and almost invariably occurs. The best demonstration of this argument, of which I am aware, is the town of Puri, where pilgrims gather at all times of the year, but in particularly large numbers to celebrate the *Rath Jatra.* I quote from my own report on the last great *mela* there.

"My own opinion as to the frequency and regularity of cholera outbreaks in Puri is that the conditions are almost ideally perfect for the occurrence of such epidemics.

The chain of necessary connecting links is complete: the climate is moist and warm: pilgrims are crowded together in large numbers: indiscriminate defœcation is almost universal: carriers of cholera infection are certain to be present in so large a population recruited from so wide an area: flies are exceedingly prevalent: and lastly, to complete the chain of circumstance, there is the "*Prasad*," the cold cooked food which the pilgrims must eat, and which they carry from one temple to another, and through the bazar, on a simple platter of leaves open to all the flies to feast on and to contaminate. This cold cooked food is an excellent medium for the multiplication of the organisms, and it is probable that many portions of this food contain large numbers of cholera organisms before they are consumed by the pilgrims, many of whom are old and most of whom have their natural resistance diminished by exposure and privation, prolonged travelling, and insufficient food."

On the other hand when any links are missing from the chain, a great *mela* may assemble and disperse without any fear of an extensive outbreak of cholera.

This condition is typically exemplified in the Sonepore *mela* at which from 2 to 4 *lakhs* of people assemble yearly in the month of *Kartik* (October-November). The crowd and the carriers are there, but the climate is not favourable to the reproduction of flies as the nights are too cold, and the conservancy is good—in fact very good—and so the flies do not find the necessary nidus : also the water-supply is clean and carefully guarded, the chances of contamination are therefore practically avoided, and the crowd and its carriers cannot alone produce an epidemic.

10. *Why Cholera is preventible in Europe.*—To elaborate the same argument further, we arrive at a working hypothesis as to why cholera is not endemic in the greater part of Europe, and why it is so easily preventible there, because during the greater part of the year the climate is unfavourable, and in most places the conservancy arrangements are really good.

11. *How Cholera is preventible in India.*—In India on the other hand the climate is more generally favourable than otherwise, but it is interesting to observe the effect of the rigorous Punjab climate in reducing the prevalence of cholera as compared with the moist and warm climates of Bengal and Madras, where cholera is endemic, whilst in Bihar and the Upper Provinces, cholera is typically a seasonal disease, becoming prevalent in the warm weather when flies can breed freely, and dying out in the rigorous cold weather of winter.

As we cannot modify the climate, it is obviously more logical to devote our energies to the improvement of conservancy, and my whole argument leads up to this—that good conservancy is the Alpha and Omega of cholera prevention,—as a practical measure, and on a large and general scale.

Crowds and crowding we cannot prevent, and the "carrier" we shall always have with us, though we may hope to decrease his numbers. Water-supplies can, and should be improved and made safer, but the essence of my argument is the necessity for breaking the chain of circumstances at its weakest and most accessible link, and fixing a great gap between man and the contamination of his food, by means of good conservancy.

ALL-INDIA SANITARY CONFERENCE—MADRAS—NOVEMBER 1912.

AN INVESTIGATION ON THE OCCURRENCE OF THE CHOLERA VIBRIO IN THE BILIARY PASSAGES.

BY

Major E. D. W. Greig, M.D., D.Sc., I.M.S., on special duty for the Cholera Enquiry.

INTRODUCTORY.

In my researches[1] on the etiology of Enteric fever in India, I dealt with the relation of the B. typhosus to the biliary passages and the important bearing which this had on the "carrier" question. When I was placed on special duty this year for the Cholera Enquiry of the Indian Research Fund Association, a problem which naturally presented itself was; is the Cholera vibrio present in or absent from the biliary passages? Turning in the first place to the most recent monograph[2] on Cholera in India (Rogers, 1911), for information on this point, I read, on page 65, the following sentence in this connection. "The absence of infection of the gall-bladder and bile ducts by the Comma bacillus places the disease in quite a different position from that of Typhoid in this respect."

Early observations on the Cholera vibrio in bile.—On referring to the literature I find that some observations had been made, before the important relationship of the occurrence to the epidemiology of Cholera was appreciated. Thus Nicati and Rietsch (1884)[3] examined the bile in 3 cases of Cholera and found the vibrio in two. On another occasion they examined 5 Cholera cases and found the vibrio in the bile in 2. Similar observations were made by Doyen (1884)[4], Tizzoni and Cantani (1886)[5], J. F. Raptschevsky (1886)[6], L. P. Rekovsky (1882)[7].

Kulescha[8] examined recently 109 Cholera cases and found the Cholera vibrio in the gall-bladder in 49, and in 10 per cent of the cases he observed changes in the biliary passages.

Brulloff L.[9] has found the Cholera vibrio in the bile *post mortem* very frequently, in 76 per cent of the cases examined, and also in the blood and other organs. Defressine C. and Cazeneuve H.[10] in November 1911 in a small epidemic of Cholera at Toulouse found the Cholera vibrio in the bile in 3 cases.

The "carrier" question is one of vital concern in regard to the prevention of Cholera and an elucidation of the exact mode of production of the "Cholera carrier" is therefore a problem of fundamental importance.

This year being the "Nutan Kalebar" Festival of Jagannath an unusually large number of pilgrims, about 300,000, visited Puri during the Car pulling. As a result of this the annual number of Cholera cases was greatly increased, and I had a very large amount of valuable material for the purpose of my research. I was enabled to systematically investigate the relationship of the Cholera vibrio to the biliary passages. I had an opportunity of making a bacteriological examination of the bile in 271 fatal cases; and the result of this investigation showed that the Cholera vibrio was found in no less than 80 of the cases; and that distinct

[1] Scientific Memoirs of the Government of India No. 32.
[2] Cholera and its treatment; Leonard Rogers, 1911, Oxford Medical publication.
[3] Recherches sor le Cholera Arch. de Physiol norm. et path Paris, 1885.
[4] Le Progress. Med. 1885, No. 27, and C. R. de la Soc. Biol. 1884 No. 42.
[5] Centralbl. f. med. Wessensch, 1886 No. 43.
[6] Wratsch, 1886 No. 45.
[7] Arch d. Scien. biol. St. Petersbourg T. I., 1892.
[8] Centralbl. f. Bakt. Abt. I Orig. Bd. 59, H. 4., 1909.
[9] Wratsch, 1910 No. 47, p. 1821.
[10] C. R. Soc. de Biolog. T, 72, 1912, p. 933.

pathological changes, both naked eye and microscopic, were found in the gall-bladder in 12 of the 80 cases; and further it is interesting to note that the Comma bacillus is found, on section of the gall-bladder, to be present not only on the surface of the mucous membrane but, also, deeper in the sub-mucous tissue. So far as I am aware my series of observations on the occurrence of the Cholera vibrio in the biliary passages is the largest recorded in the literature.

Before proceeding to describe my research in detail it may be well to direct attention to the importance of this observation in relation to the causation and prevention of Cholera.

If the Cholera vibrio is an inhabitant of the intestine only and does not gain access to the biliary passages as stated[1] in a standard text book of bacteriology, a view which is very generally held at present, then the conditions for the prolonged life of the organism in the body of the host after recovery from the acute attack would be much less favourable, as the delicate Comma bacillus would have to enter in a struggle with the other intestinal and putrefactive organisms, and that too in an unfavourable medium, and although an "acute or temporary carrier" might result, the possibility of a "permanent Chronic Cholera carrier" being produced in these circumstances would be slight, on the other hand if it can enter the gall-bladder it finds there ideal conditions for its prolonged life, namely, the absence of other competitors and a suitable alkaline medium, indeed Ottolenghi[2] has recently recommended bile as a selective medium for "enriching" the Comma bacillus in place of peptone water, and I can confirm the value of this medium from my experience. My researches have demonstrated that the Cholera vibrio can enter and live in the bile, and the fact that Cholera vibrio enters and lives in the bile increases the chances of the production of the "chronic Cholera carrier." From the point of view of the prevention of Cholera the "chronic carrier" is a much more serious problem to deal with than an "acute carrier" although the latter is by no means without significance and both deserve close consideration. An apparently healthy person, whether convalescent or "contact" harbouring the Cholera vibrio in the gall-bladder, is dangerous in a high degree, because he is liable to start fresh foci of infection, it may be in various widely separated places to which he may travel, and, possibly, retaining in his body the organism of Cholera for prolonged periods. Such a person acts as a reservoir of the virus.

Owing to the fact that the recovery from cholera is remarkably fast and that the patients are, as a rule, discharged at the first opportunity from hospital, there is much greater danger in this disease of highly infective persons returning to the community than in enteric fever, in which the convalescence is slower. In another communication I shall deal with the question of cholera convalescents.

Methods.

As soon as possible after the death of the cholera patient I removed the gall-bladder having previously ligatured the common bile duct. The gall-bladders were sent to my temporary laboratory at Puri and the following technique for their investigation was adopted. A small part of the outside of the gall-bladder was seared with a hot glass rod, and through this sterile patch a sterile glass pipette was passed and the bile drawn up and transferred to suitable media. After this operation the gall-bladder was opened and the condition of the contents and wall noted, if there was any sign of pathological alteration in the wall, a portion was taken, fixed and hardened for sectioning in paraffin. For the bacteriological investigation I used at first, (*a*) ordinary agar slopes, (*b*) agar plates with preliminary "enrichment" in peptone water, (*c*) plating on Dieudonné medium, but as the observations went on I found that equally good results were obtained by the first method (*a*), *i.e.*, transference of a small quantity of bile to agar slopes with a sterile pipette, as with the other two methods, so that latterly I confined myself to method (*a*) only. The culture was in each case tested with a higher titre cholera agglutinating serum, one prepared by myself by treating

[1] Manual of Bacteriology. Muir and Ritchie, 1910.

[2] Centralbl f. Bakt. B. D. 58. H. 4., 1911.

rabbits intravenously with increasing doses of a living agar culture of a cholera vibrio obtained from a fatal case in Calcutta. The titre of my serum was 1-8,000. I also used as a control a high titre cholera agglutinating serum (1-10,000) obtained from the Swiss Serum and Vaccine Institute, Berne, prepared under the supervision of Professor Kolle. With a low dilution of these sera (1-50) immediate clumping, observed microscopically, occurred when the cholera vibrio culture was mixed with a small quantity of the serum dilution on a slide, and when tested subsequently the vibrios agglutinated to the end point of the high titre serum. Each culture was examined also microscopically, both stained and unstained, to determine whether or not the organisms had the characters of the cholera vibrio. The appearance on various other culture media was noted also. The identity of each strain was thus fully established by microscopic, cultural, and serological tests.

Tissue for examination was fixed in alcohol and prepared and embedded in paraffin in the usual manner. Serial sections were cut and stained in carbol fuchsin and in carbol thionine. Drawings were prepared to show the details of the structural changes.

Record of observations.

The following table shows the sex, caste, the date of death, date of examination of the gall-bladder, the condition of the bile and the gall-bladder, the presence or absence of the cholera vibrio in the bile of fatal cases of cholera :—

No.	Sex.	Caste.	Date of death.	Date of removal of gall-bladder and examination of bile.	Condition of the bile and the gall-bladder.	Presence or absence of cholera vibrio in bile.
1	F.	Hindu pilgrim...	20th July 1912 ...	20th July 1912 ...	Dark ; normal	+
2	M.	Ditto ...	Ditto ...	Ditto ...	Yellow ; normal	+
3	F.	Ditto ...	Ditto ...	Ditto ...	Dark ; normal	— (Sterile.)
4	F.	Ditto ...	Ditto ...	Ditto ...	Ditto	— Do.
5	F.	Ditto ...	Ditto ...	Ditto ...	Ditto	— Do.
6	F.	Ditto ...	Ditto ...	Ditto ...	Yellow ; normal	— Do.
7	M.	Ditto ...	Ditto ...	Ditto ...	Dark ; normal	— Do.
8	M.	Ditto ...	21st July 1912 ...	21st July 1912 ...	Dark, fluid ; normal	— Do.
9	M.	Ditto ...	Ditto ...	Ditto ...	Dark green, viscid, large gall stones ...	+
10	F.	Ditto ...	Ditto ...	Ditto ...	Dark, green ; healthy	— Do.
11	F.	Ditto ...	Ditto ...	Ditto ...	Ditto	+
12	M.	Ditto ...	Ditto ...	Ditto ...	Dark brown ; fluid ; bile strained ...	+
13	F.	Ditto ...	Ditto ...	Ditto ...	Dark fluid ; healthy	+
14	M.	Ditto ...	Ditto ...	Ditto ...	Dark viscid ; healthy	— (Qther col.)
15	M.	Ditto ...	Ditto ...	Ditto ...	Light green fluid ; healthy	— Do.
16	M.	Ditto ...	Ditto ...	Ditto ...	Yellow fluid ; healthy	+
17	F.	Ditto ...	Ditto ...	Ditto ...	Dark viscid ; healthy	+
18	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	— (Sterile.)
19	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	— Do.
20	M.	Ditto ...	Ditto ...	Ditto ...	Dark brown ; healthy	— Do.
21	M.	Ditto ...	Ditto ...	Ditto ...	Dark green fluid ; normal	— Do.
22	F.	Ditto ...	Ditto ...	Ditto ...	Dark viscid ; normal	— Do.
23	M.	Ditto ...	Ditto ...	Ditto ...	Dark fluid ; normal	+
24	F.	Ditto ...	Ditto ...	Ditto ...	Dark brown ; normal ; pigmented	+
25	M.	Ditto ...	Ditto ...	Ditto ...	Dark tarry ; normal	— (Other col.)
26	F.	Ditto ...	Ditto ...	Ditto ...	Ditto	— (Sterile.)
27	M.	Ditto ...	Ditto ...	Ditto ...	Dark brown ; normal	— Do.
28	F.	Ditto ...	Ditto ...	Ditto ...	Dark ; normal ; pigmented	— Do.
29	M.	Ditto ...	22nd July 1912 ...	22nd July 1912 ...	Dark fluid ; healthy	— Do.

No.	Sex.	Caste.	Date of death.	Date of removal of gall-bladder and examination of bile.	Condition of the bile and the gall-bladder.	Presence or absence of cholera vibrio in bile.
30	F.	Hindu pilgrim	22nd July 1912 ...	22nd July 1912 ...	Very dark fluid; healthy...	— (Sterile.)
31	M.	Ditto ...	Ditto ...	Ditto ...	Thick dark; healthy, pigmented	— Do.
32	F.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; healthy	— Do.
33	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	— Do.
34	M.	Ditto ...	Ditto ...	Ditto ...	Dark green viscid; healthy, pigmented ...	— Do.
35	F.	Ditto ...	Ditto ...	Ditto ...	Dark green fluid; healthy	+
36	F.	Ditto ...	Ditto ...	Ditto ...	Ditto	— Do.
37	M.	Ditto ...	Ditto ...	Ditto ...	Very dark tarry; healthy	— Do.
38	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	+
39	F.	Ditto ...	Ditto ...	Ditto ...	Dark fluid; healthy	— Do.
40	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	— Do.
41	F.	Ditto ...	Ditto ...	Ditto ...	Dark brown fluid; healthy	— Do.
42	M.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; healthy	+
43	F.	Ditto ...	Ditto ...	Ditto ...	Ditto	— Do.
44	M.	Ditto ...	Ditto ...	Ditto ...	Dark fluid; healthy	— Do.
45	F.	Ditto ...	Ditto ...	Ditto ...	Fluid; healthy	— Do.
46	M.	Ditto ..	Ditto ...	Ditto ...	Dark tarry; healthy	+
47	F.	Ditto ...	Ditto ...	Ditto ...	Fluid dark; healthy	— Do.
48	M.	Ditto ...	Ditto ...	Ditto ...	Dark viscid; healthy, pigmented	— Do.
49	F.	Ditto ...	Ditto ...	Ditto ...	Tarry bile; healthy	— Do.
50	M.	Ditto ...	23rd July 1912 ...	23rd July 1912 ...	Dark green fluid bile; thickened	— Do.
51	M.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; normal	— Do.
52	F.	Ditto ...	Ditto ...	Ditto ...	Ditto	+
53	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	— Do.
54	F.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; healthy	— Do.
55	M.	Ditto ...	Ditto ...	Ditto ...	Dark brown; healthy	— Do.
56	F.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; healthy	+
57	F.	Ditto ...	Ditto ...	Ditto ...	Very little bile; shrunk and small	— Do.
58	M.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; healthy	— Do.
59	F.	Ditto ...	Ditto ...	Ditto ...	Ditto	— Do.
60	M.	Ditto ...	Ditto ...	Ditto ...	Dark; healthy	— Do.
61	F.	Ditto ...	Ditto ...	Ditto ...	Dark brown; healthy	— Do.
62	M.	Ditto ...	Ditto ...	Ditto ...	Dark fluid; healthy	+
63	M.	Ditto ...	Ditto ...	Ditto ...	Dark green viscid; healthy	— Do.
64	M.	Ditto ...	Ditto ...	Ditto ...	Fluid green; healthy	— Do.
65	M.	Ditto ...	Ditto ...	Ditto ...	Dark fluid; wall healthy	+
66	M.	Ditto ...	24th July 1912 ...	24th July 1912 ...	Thick tarry; healthy	+
67	M.	Ditto ...	Ditto ...	Ditto ...	Dark green; viscid; healthy	+
68	M.	Ditto ...	Ditto ...	Ditto ...	Tarry; healthy	— Do.
69	M.	Ditto ...	Ditto ...	Ditto ...	Yellowish green fluid; healthy	— Do.
70	F.	Ditto ...	Ditto ...	Ditto ...	Dark green fluid; healthy	— Do.
71	F.	Ditto ...	Ditto ...	Ditto ...	Dark fluid; healthy	— Do.
72	F.	Ditto ...	Ditto ...	Ditto ...	Dark brown viscid; healthy	— Do.
73	M.	Ditto ...	Ditto ...	Ditto ...	Dark brown; healthy	— Do.
74	M.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; healthy	+
75	M.	Ditto ...	Ditto ...	Ditto ...	Dark fluid; healthy	— Do.
76	M.	Ditto ...	Ditto ...	Ditto ...	Green fluid; healthy	+

No.	Sex.	Caste.	Date of death.	Date of removal of gall-bladder and examination of the bile.	Condition of the bile and the gall-bladder.	Presence or absence of cholera vibrio in bile.
77	M.	Hindu pilgrim	24th July 1912 ...	24th July 1912 ...	Enlarged 7 × 2½ thin (full of dark bile) healthy	—
78	F.	Ditto ...	Ditto ...	Ditto ...	Dark green fluid; healthy	+
79	F.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; healthy	+
80	F.	Ditto ...	Ditto ...	Ditto ...	Dark fluid; healthy	—
81	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	+
82	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	— (Sterile.)
83	M.	Ditto ...	Ditto ...	Ditto ...	Clear yellowish slightly fluid; congested ...	— Do.
84	M.	Ditto ...	Ditto ...	Ditto ...	Dark fluid; healthy	— Do.
85	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	+
86	M.	Ditto ...	Ditto ...	Ditto ...	Small contracted; wall congested and adherent to liver somewhat thickened contains light brown fluid.	— Do.
87	F.	Ditto ...	25th July 1912 ...	25th July 1912 ...	Dark tarry; healthy	— Do.
88	F.	Ditto ...	Ditto ...	Ditto ...	Dark fluid; healthy	+
89	F.	Ditto ...	Ditto ...	Ditto ...	Ditto	— Do.
90	F.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; healthy	+
91	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	— Do.
92	M.	Ditto ...	Ditto ...	Ditto ...	Thick tarry; healthy	— Do.
93	M.	Ditto ...	Ditto ...	Ditto ...	Distended with light brown bile healthy ...	— Do.
94	M.	Ditto ...	Ditto ...	Ditto ...	Dark green fluid; healthy ...	— Do.
95	M.	Ditto ...	Ditto ...	Ditto ...	Light brown fluid; three gall stones ...	— Do.
96	M.	Ditto ...	Ditto ...	Ditto ...	Thick tarry; healthy	— Do.
97	M.	Ditto ...	Ditto ...	Ditto ...	Green tarry; healthy	+
98	M.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; healthy	— Do.
99	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	— Do.
100	F.	Ditto ...	Ditto ...	Ditto ...	Filled up with two large gall stones; bile escaped in removing it.	— Do.
101	F.	Ditto ...	Ditto ...	Ditto ...	Green fluid; healthy	+
102	F.	Ditto ...	Ditto ...	Ditto ...	Thin fluid bile; healthy	+
103	M.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; healthy	— Do.
104	M.	Ditto ...	Ditto ...	Ditto ...	Dark brown; healthy	+ Do.
105	M.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; healthy	— Do.
106	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	— Do.
107	M.	Ditto ...	Ditto ...	Ditto ...	Dark brown; healthy	— Do.
108	M.	Ditto ...	Ditto ...	Ditto ...	Very thick tarry; healthy	— Do.
109	M.	Ditto ...	26th July 1912 ...	26th July 1912 ...	Dark green; healthy	— Do.
110	M.	Ditto ...	Ditto ...	Ditto ...	Dark fluid; healthy	+
111	F.	Ditto ...	Ditto ...	Ditto ...	Dark brown; healthy	— Do.
112	F.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; healthy	+
113	F.	Ditto ...	Ditto ...	Ditto ...	Ditto	— Do.
114	M.	Ditto ...	Ditto ...	Ditto ...	Yellow; wall intensely congested throughout ...	+
115	M.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; wall healthy	— Do.
116	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	+
117	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	— Do.
118	F.	Ditto ...	Ditto ...	Ditto ...	Yellow green; wall congested in patches ...	+
119	F.	Ditto ...	Ditto ...	Ditto ...	Tarry black; wall healthy	— Do.
120	F.	Ditto ...	Ditto ...	Ditto ...	Ditto	+
121	F.	Ditto ...	27th July 1912 ...	27th July 1912 ...	Yellow; healthy	— Do.

No.	Sex.	Caste.	Date of death.	Date of removal of gall-bladder and examination of bile.	Condition of the bile and gall-bladder.	Presence or absence of cholera vibrio in bile.
122	F.	Hindu pilgrim...	27th July 1912 ...	27th July 1912 ...	Dark tarry; healthy	+
123	F.	Ditto ...	Ditto ...	Ditto ...	Ditto	— (Sterile.)
124	F.	Ditto ...	Ditto ...	Ditto ...	Brown liquid; healthy	— Do.
125	F.	Ditto ...	Ditto ...	Ditto ...	Brown viscid; healthy	— Do.
126	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	+
127	M.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; healthy	— Do.
128	M.	Ditto ...	Ditto ...	Ditto ...	Dark green; healthy	— Do.
129	M.	Ditto ...	Ditto ...	Ditto ...	Brown fluid; healthy	— Do.
130	M.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; healthy	— Do.
131	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	— (Other col.)
132	M.	Ditto ...	Ditto ...	Ditto ...	Dark brown; healthy	— (Sterile.)
133	F.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; healthy	+
134	F.	Ditto ...	Ditto ...	Ditto ...	Dark brown; healthy	+
135	F.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; healthy	— (Sterile.)
136	M.	Ditto ...	28th July 1912 ...	28th July 1912 ...	Ditto	— Do.
137	M.	Ditto ...	Ditto ...	Ditto ...	Dark brown, tarry; healthy	— Do.
138	M.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; healthy	— Do.
139	M.	Ditto ...	Ditto ...	Ditto ...	Dark green tarry; healthy	— Do.
140	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	— Do.
141	F.	Ditto ...	Ditto ...	Ditto ...	Ditto	— Do.
142	M.	Ditto ...	29th July 1912 ...	29th July 1912 ...	Dark tarry; healthy	— Do.
143	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	— Do.
144	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	— Do.
145	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	+
146	M.	Ditto ...	Ditto ...	Ditto ...	Dark green; healthy	— Do.
147	M.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; healthy	— Do.
148	M.	Ditto ...	Ditto ...	Ditto ...	Dark yellow; healthy	— Do.
149	M.	Ditto ...	Ditto ...	Ditto ...	Dark brown; healthy	+
150	F.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; healthy	+
151	F.	Ditto ...	Ditto ...	Ditto ...	Dark green; healthy	+
152	F.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; healthy	+
153	F.	Ditto ...	Ditto ...	Ditto ...	Dark fluid; healthy	— Do.
154	F.	Ditto ...	Ditto ...	Ditto ...	Yellow fluid; inflamed	+
155	F.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; healthy	— (Other col.)
156	F.	Ditto ...	Ditto ...	Ditto ...	Very dark tarry; healthy	— (Sterile.)
157	M.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; healthy	—
158	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	—
159	M.	Ditto ...	Ditto ...	Ditto ...	Dark green; healthy	—
160	M.	Ditto ...	31st July 1912 ...	31st July 1912 ...	Dark brown fluid; wall greatly thickened and inflamed.	+
161	M.	Ditto ...	Ditto ...	Ditto ...	Pale green very fluid; 2 gall stones; wall healthy	+
162	M.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; healthy	— (Other col.)
163	F.	Ditto ...	Ditto ...	Ditto ...	Ditto	—
164	M.	Ditto ...	Ditto ...	Ditto ...	Dark green fluid; healthy	+ (Sterile.)
165	F.	Ditto ...	Ditto ...	Ditto ...	Dark tarry bile; healthy	— Do.
166	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	+
167	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	— Do.
168	M.	Ditto ...	Ditto ...	Ditto ...	Yellowish fluid; healthy	— (Other col.)

No.	Sex.	Caste.	Date of death.	Date of removal of gall-bladder and examination of bile.	Condition of the bile and the gall-bladder.	Presence or absence of cholera vibrio in bile.
169	M.	Hindu pilgrim...	1st August 1912 ...	1st August 1912 ...	Brown fluid bile; healthy	— (Other col.)
170	M.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; healthy	— (Sterile.)
171	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	+
172	M.	Ditto ...	Ditto ...	Ditto ...	Dark green fluid; healthy	+
173	M.	Ditto ...	Ditto ...	Ditto ...	Viscid green bile; healthy	+
174	F.	Ditto ...	Ditto ...	Ditto ...	Brown fluid; congested	— Do.
175	M.	Ditto ...	Ditto ...	Ditto ...	Dark green viscid; healthy	+
176	F.	Ditto ...	2nd August 1912 ...	2nd August 1912 ...	Dark brown fluid; healthy	+
177	F.	Ditto ...	Ditto ...	Ditto ...	Green fluid; healthy	+
178	M.	Ditto ...	Ditto ...	Ditto ...	Dark tarry bile; healthy	— Do.
179	F.	Ditto ...	Ditto ...	Ditto ...	Yellow viscid (somewhat); intensely congested...	+
180	F.	Ditto ...	3rd August 1912 ...	3rd August 1912 ...	Dark tarry; healthy	+
181	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	— (Sterile.)
182	M.	Ditto ...	4th August 1912 ...	4th August 1912 ...	Dark brown viscid; healthy	— Do.
183	M.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; healthy	— Do.
184	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	— Do.
185	M.	Ditto ...	Ditto ...	Ditto ...	Dark brown viscid; healthy	+
186	F.	Ditto ...	5th August 1912 ...	5th August 1912 ...	Green fluid; healthy	+
187	M.	Ditto ...	Ditto ...	Ditto ...	Brown fluid; healthy	— (Sterile.)
188	M.	Ditto ...	6th August 1912 ...	6th August 1912 ...	Dark tarry; healthy	— Do.
189	M.	Ditto ...	Ditto ...	Ditto ...	Dark fluid bile; healthy ...	— Do.
190	F.	Ditto ...	Ditto ...	Ditto ...	Dark green bile; congested	— Do.
191	F.	Ditto ...	Ditto ...	Ditto ...	Dark colour bile; healthy	— Do.
192	M.	Ditto ...	7th August 1912 ...	7th August 1912 ...	Dark brown; healthy	— Do.
193	M.	Ditto ...	Ditto ...	Ditto ...	Dark fluid; healthy	— Do.
194	M.	Ditto ...	Ditto ...	Ditto ...	Tarry bile; healthy	— Do.
195	M.	Ditto ...	8th August 1912 ..	8th August 1912 ...	Dark tarry bile; healthy	— Do.
196	F.	Ditto ...	9th August 1912 ...	9th August 1912 ...	Fluid dark; healthy	— Do.
197	F.	Ditto ...	Ditto ...	Ditto ...	Dark brown; healthy	— Do.
198	M.	Ditto ...	Ditto ...	Ditto ...	Dark fluid; healthy	— Do.
199	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	— Do.
200	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	— Do.
201	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	+
202	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	— (Other col.)
203	M.	Ditto ...	Ditto ...	Ditto ...	14 gall stones; healthy	— (Sterile.)
204	M.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; healthy	— (Other col.)
205	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	— (Sterile.)
206	F.	Ditto ...	Ditto ...	Ditto ...	Ditto	— Do.
207	M.	Ditto ...	10th August 1912 ...	10th August 1912 ...	Fluid bile; healthy	— Do.
208	M.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; healthy	+
209	M.	Ditto ...	Ditto ...	Ditto ...	Fluid; healthy	— Do.
210	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	— Do.
211	M.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; healthy	— Do.
212	M.	Ditto ...	11th August 1912 ...	11th August 1912 ...	Dark green; healthy	— (Other col.)
213	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	— (Sterile.)
214	...	Ditto ...	Ditto ...	Ditto ...	Ditto	— Do.
215	...	Ditto ...	Ditto ...	Ditto ...	Ditto	— Do.
216	...	Ditto ...	Ditto ...	Ditto ...	Ditto	+

No.	Sex.	Caste.	Date of death.	Date of removal of gall-bladder and examination of bile.	Condition of the bile and the gall-bladder.	Presence or absence of cholera vibrio in bile.
217	...	Hindu pilgrim ...	11th August 1912 ...	11th August 1912 ...	.....	+
218	F.	Ditto ...	12th August 1912 ...	12th August 1912 ...	Green fluid; healthy	— (Sterile.)
219	F.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; healthy	+
220	F.	Ditto ...	Ditto ...	Ditto ...	Ditto	— Do.
221	M.	Ditto ...	Ditto ...	Ditto ...	Dark brown; healthy	— Do.
222	F.	Ditto ...	13th August 1912 ...	13th August 1912 ...	Dark green fluid; healthy	— Do.
223	F.	Ditto ...	Ditto ...	Ditto ...	Ditto	— (Other col.)
224	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	+
225	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	+
226	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	— (Sterile.)
227	M.	Ditto ...	Ditto ...	Ditto ...	Dark tarry viscid; healthy	— Do.
228	M.	Ditto ...	15th August 1912 ...	15th August 1912 ...	Dark fluid; healthy	— Do.
229	M.	Ditto ...	Ditto ...	Ditto ...	Dark viscid; healthy	— Do.
230	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	— Do.
231	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	— Do.
232	M.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; healthy	— Do.
233	M.	Ditto ...	Ditto ...	Ditto ...	Dark brown; slightly congested	+
234	M.	Ditto ...	16th August 1912 ...	16th August 1912 ...	Dark fluid; healthy	— (Other col.)
235	M.	Ditto	Ditto ...	Ditto ...	Dark tarry; healthy	— (Sterile.)
236	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	— (Other col.)
237	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	— Do.
238	M.	Ditto ...	Ditto ...	Ditto ...	Dark fluid brown; healthy	— Do.
239	M.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; healthy	— Do.
240	M.	Ditto ...	Ditto ...	Ditto ...	.....	— (Sterile.)
241	M.	Ditto ...	Ditto ...	Ditto ...	.....	— Do.
242	M.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; healthy	— Do.
243	F.	Ditto ...	17th August 1912 ...	17th August 1912 ...	Ditto	— Do.
244	M.	Ditto ...	Ditto ...	Ditto ...	Dark green fluid; healthy	— Do.
245	M.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; healthy	+
246	F.	Ditto ...	18th August 1912 ...	18th August 1912 ...	Ditto	— Do.
247	F.	Ditto ...	Ditto ...	Ditto ...	Yellowish brown; healthy	— (Other col.)
248	M.	Ditto ...	Ditto ...	Ditto ...	Dark tarry; healthy	— (Sterile.)
249	M.	Ditto ...	19th August 1912 ...	19th August 1912 ...	Fluid brown; healthy	+
250	F.	Ditto ...	Ditto ...	Ditto ...	Green fluid; healthy	—(Other col.).
251	M.	Ditto ...	Ditto ...	Ditto ...	Brown fluid; congested	— Do.
252	M.	Ditto ...	20th August 1912 ...	20th August 1912 ...	Tarry; healthy	—(Sterile.)
253	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	— Do.
254	F.	Ditto ...	Ditto ...	Ditto ...	Dark green fluid; congested	+
255	M.	Ditto ...	21st August 1912 ...	21st August 1912 ...	Liquid brown; healthy	—(Other col.)
256	M.	Ditto ...	Ditto ...	Ditto ...	Dark tarry bile; healthy	+
257	M.	Ditto ...	Ditto ...	Ditto ...	Dark fluid; healthy	+
258	F.	Ditto ...	Ditto ...	Ditto ...	Dark brown; healthy	—(Sterile.)
259	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	— Do.
260	F.	Ditto ...	Ditto ...	Ditto ...	Ditto	— Do.
261	M.	Ditto ...	Ditto ...	Ditto ...	Ditto	+
262	F.	Ditto ...	22nd August 1912 ...	22nd August 1912 ...	Dark green; healthy	— Do.
263	M.	Ditto ...	23rd August 1912 ...	23rd August 1912 ...	Ditto	+

No.	Sex.	Caste.	Date of death.	Date of removal of gall-bladder and examination of bile.	Condition of the bile and the gall-bladder.	Presence or absence of cholera vibrio in bile.
264	F.	Hindu pilgrim ..	25th August 1912 ...	25th August 1912 ...	Light green; injected	+
265	M.	Ditto ...	Ditto ...	Ditto ...	Dark green fluid; healthy	—(Sterile.)
266	F.	Ditto ...	27th August 1912 ...	27th August 1912 ...	Dark brown; healthy	—(Other col.)
267	M.	Ditto ...	28th August 1912 ...	28th August 1912 ...	Dark tarry; healthy	+
268	M.	Ditto ...	30th August 1912 ...	30th August 1912 ...	Yellowish brown; healthy	— Do.
269	M.	Ditto ...	Ditto ...	Ditto ...	Intensely inflamed	+
270	M.	Ditto ...	1st September 1912.	1st September 1912.	Liquid greenish bile; wall healthy ...	—(Sterile.)
271	F.	Ditto ...	4th September 1912.	5th September 1912...	Intensely inflamed	+

From these results it will be seen that the Cholera vibrio can enter and develop in the bile. In the great majority of cases in which the comma bacillus was determined it was present in large numbers and in pure culture in the bile, showing that it had found a particularly favourable medium for its growth. It is obvious that the bile containing the Cholera vibrio in large numbers will be poured out into the small intestine and the organisms will gain an exit to the outside world with the faeces. I have determined the presence of the Cholera vibrio in the stool of a person who was quite healthy at the time but had passed through an attack of Cholera some weeks previously. In examining a number of cholera convalescents daily I have noted[1] that the discharge of the vibrios may be intermittent as in the case of B. typhosus. Zlalogoroff[2] has found the cholera vibrio in the stool one year after the attack.

These observations have a most important relation to the epidemiology of Cholera because they afford a rational explanation in harmony with recorded facts of the mode of production of the "chronic carrier."

In 68 cases no pathological change was observed in the biliary passages although the comma bacillus was present in pure culture; like the B. typhosus the Cholera vibrio can live in the biliary system without producing a pathological reaction, although, as we shall see, this is not invariably the case, and interesting pathological changes are found in the gall-bladder in some cases. In typhoid fever the B. typhosus after circulating in the blood stream for a time, deposits locally in various tissues and the B. typhosus appears to gain access to the biliary passages from the blood stream. In Cholera it may be that the entrance is from the intestine, Cholera vibrios being very abundant in the (rice water) contents of the small intestine, differing in this respect from the B. typhosus; the observations of Brulloff,[2] however, already referred to, who found the cholera vibrio in the blood, and, also, my own observation, dealt with later in this paper, that the comma bacillus may occur in the lung, have to be remembered; and it may be shown that the cholera vibrio like the B. typhosus gains an entrance to the bile by the blood stream. Baroni V and Ceaparie Victoria[3] injected large quantities of cholera vibrios into the ear vein of a rabbit and they found them in the bile 30 minutes after injection but not in the small intestine till one hour after. In small doses the vibrios rapidly disappear from the circulation and deposit in the small intestine, appendix, and the bile. In the urine the vibrio was never found.

It is interesting to note that the bile, from cases of cholera, in which the vibrio was found, is, as a rule, sterile—163 cases out of a total of 190 cases;

[1] An investigation of Cholera Convalescents and contacts by Major E. D. W. Greig.

[2] Centralbl. f. Bact., Vol., 52 H. 2 1909.

[3] C. R. Soc. de Biol. T. 72 1912, page 894.

of the remaining 27 cases in which the bile was found to contain other organisms a certain number may be accounted for by *post mortem* invasion of the bile. The bile, being generally sterile, will for this reason be a very suitable habitat for the delicate comma bacillus, as there are no other organisms to interfere with its growth, such as it would meet with in the intestine. It will thus be "protected" from other competitors.

Pathological changes in the gall-bladder in Cholera cases.—In my previous researches on enteric fever in India, 1906, I noted that the B. typhosus could give rise to cholecystitis. Forster[2] of Strassburg was the first to associate gall-bladder complaints with the "carrier" in typhoid fever. Ledingham[3], Droba (1899)[4], Miller (1899)[5], Brion (1910)[6], Findlay and Buchanan (1906)[7], Simion (1907)[8], Dudgeon (1908)[9], and other observers have recorded cases in connection with typhoid fever, but the relationship of the cholera vibrio to pathological lesions of the gall-bladder has received very little attention. Yet, both in view of the elucidation of the "carrier" question and the possible causal connection of gall-bladder infection with the various toxic sequelæ met with in cholera, the subject is one of great practical importance. I have already referred to the work of Kulescha[10] on the gall-bladder in cholera. He records an interesting case not recognised as cholera during life, but at the *post mortem* examination the biliary passages in the liver were found to be greatly distended and filled with pus from which the cholera vibrio was isolated. Sections showed that there were other organisms present so that it could not be regarded as a pure vibrio infection.

During convalescence from cholera in a few cases I have observed that pain has been complained of in the right hypochondrium; and on palpation a tender swelling, having the shape and position of the gall-bladder has been felt. This swelling subsided in a few days in each case and the patients recovered.

In a case recorded by me[11] in which the patient lived for 12 days after recovery from the acute attack of cholera and finally died of uraemia, the gall-bladder was found to be intensely congested and the bile contained a pure culture of the comma bacillus. It is possible that the common fatal complication of cholera, uraemia, is brought about in certain cases at least by the continuance of an active focus of infection in the gall-bladder, toxine being absorbed from it into the general circulation and giving rise to lesions of the kidneys and other organs.

A point of special interest in the case above referred to was that patches of consolidation were present in the right lung and that smear preparations and sections made from these areas showed the presence of a comma bacillus, and it must have gained access to the lungs by the blood stream, and the question arises therefore whether or not cholera is to be regarded as a septicaemia rather than a pure toxaemia. Including this case I found distinct pathological changes in the gall-bladder in 12 cases in my series in which the cholera vibrio was present in pure culture in the bile.

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[1] Scientific Memoirs, Government of India, No. 32.

[2] Muench. Med. Woch. 1905, page 1473.

[3] Reports L. G. B. New Series No. 43.

[4] Droba-Wien. Klin. Woch. 1899, page 1651.

[5] Bull. Johns. Hop. Hosp. Vol. 9, page 95.

[6] Cent. f. Bakt. Abt. I. Orig. Bd. 30.

[7] Glasg. Med. Jour. Vol. 65, page 177.

[8] Klin. Jahrbuch Bd. 17, page 363.

[9] Lancet, 1908, Vol. 2, page 1651.

[10] *Ibid.*

[11] In the press.

Since recording the above case I have had an opportunity of studying in detail another case of cholera in which the gall-bladder showed marked pathological changes and the bile contained a pure culture of the Cholera vibrio. It may be well to give a short history of this case here :—

Name—Champa, Hindu, Female, age 55, admitted on 4th September 1912 at 1-30 P.M., Died 9 P.M.

State on admission.—Patient was brought to hospital in an unconscious condition. She had been attacked 3 days previously. Stools typical (rice water) vomitting and purging. Pulse imperceptible. She was injected intravenously immediately after arrival with 4 pints of hypertonic salt solution. The pulse became perceptible after transfusion. Permanganate was given by the mouth. At about 5 P.M. the pulse again became imperceptible and another intravenous injection of hypertonic salt solution was given, but the pulse did not improve. Rectal injections of normal salines were given every 2 hours.

Post mortem examination.—5th September 1912.

External appearance.—Body fairly well nourished.

No increase of fluid in pleural or pericardial cavities.

Right Lung.—Weight 12 oz. Somewhat congested, especially the lower lobe, otherwise no noteworthy change.

Left Lung.—Weight 12 oz. No noteworthy change.

Heart.—Weight 6 oz. There is some hypertrophy of the wall of the left ventricle, otherwise no noteworthy change.

No increase of peritoneal fluid.

Liver.—Weight 2 lb. 2 oz. It is congested and somewhat friable.

Gall-bladder.—It is very small and shrunked ; it was removed after ligature of the duct and a portion of the surface was seared with a hot rod, and a pipette passed through the wall at the seared patch and some of the contents drawn off and inseminated on an agar slope. Next day this was found to contain a pure growth of cholera vibrio. On opening the gall-bladder there was a small quantity of dirty brown coloured bile. The mucous membrane was intensely congested and showed areas of haemorrhage in the submucosa.

Right Kidney.—Wieght 4 oz. Capsule is distinctly adherent and the substance tears on stripping it. Surface has a mottled pale grey appearance.

On section.—The cortex is pale grey with red points; pyramids pale red in colour.

Left Kidney.—Weight 5 oz. General condition practically the same as the right.

Spleen.—Normal.

Stomach and intestines.—Contents of small intestine watery.

Remarks.—In this case, also, the gall-bladder showed marked pathological change and the bile contained a pure culture of the cholera vibrio. The patient was a female of 55 years of age and the kidney showed the appearances of a lesion of old standing and the heart showed some hypertrophy of the wall of the left ventricle. The toxine of cholera absorbed both from the intestine and gall-bladder acted on the already damaged organs with the result that the first rally was followed by a second and fatal collapse. The patient died on the fourth day after the attack. The toxine in the intestine may have been destroyed by the permanganate, but the active vibrios in the gall-bladder would not be influenced by the drug and probably produced the second fatal collapse.

This condition of inflammation of the gall-bladder with a pure culture of the cholera vibrio in the bile is one of very considerable importance, as it may explain some of the severe toxic complications met with in cholera after the acute collapse stage has been recovered from. As the toxine centre in the gall-bladder will not be affected by drugs, such as permanganate, given by the

mouth with the object of destroying the toxine of cholera in the intestine, further research on the problem of killing the cholera vibrio in the tissue of the human host is required. The recent advances in Chemo-therapy of Ehrlich, Morgenroth, and others, encourage the hope that it may be possible to obtain a chemical substance capable of destroying the Cholera vibrio without damaging the tissues of the host too severely. If such a drug were obtained it would be undoubtedly of enormous help in preventing the spread of cholera. Perhaps in the mean-time serum of vaccine therapy may afford a means of dealing with the vibrios in the tissues.

The occurrence of the comma bacilli in the consolidated area of a lung in one of the cases in which the gall-bladder was intensely inflamed and the bile contained a pure culture of the cholera vibrio, was interesting and suggestive. It must have gained access to this point *viâ* the blood stream. From the point of view of prevention of cholera, the occurrence of the cholera vibrio in the consolidated area of the lung is of importance because it may gain an exit in the sputum to the external world and the possibility of this occurrence must be remembered in disinfecting the discharges of the patient.

ALL-INDIA SANITARY CONFERENCE—MADRAS—NOVEMBER 1912.

AN INVESTIGATION OF CHOLERA CONVALESCENTS AND CONTACTS IN INDIA

BY

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In my research on Enteric fever in India,* I made a daily bacteriological examination of the stools of a number of convalescent Enteric fever cases.

During the present enquiry on Cholera I was able to investigate the "carrier" problem in connection with the disease, and had an opportunity of examining stools of Cholera convalescents and contacts. During the Nutan Kalebar festival of Jagannath at Puri this year, it was estimated roughly that 300,000 pilgrims assembled at Puri during the Car pulling in July 1912, and, consequently, the annual epidemic of Cholera was larger than in previous years, and this epidemic afforded valuable material for studying various problems connected with Cholera.

The following table shows the monthly admission to the Cholera Hospital, Puri, from 1906 to 1912 (up to August) :—

Month.	1906.	1907.	1908.	1909.	1910.	1911.	1912.
January	9	115	8	2	31	7	11
February	30	25	20	9	4	8	8
March	14	9	37	45	13	5	27
April	2	3	141	16	1	4	3
May	5	*Nil*	10	3	*Nil*	8	11
June	30	2	58	274	3	31	16
July	14	130	14	14	58	28	417
August	15	23	4	2	18	3	155
September	12	6	7	3	6	8	...
October	9	15	13	6	14	22	...
November	22	37	7	4	11	6	...
December	5	2	*Nil*	3	3	*Nil*	...

It will be seen from this table that the number of admissions for Cholera during this Car Festival (1912) was much higher than in previous years. The festival is held annually in June or July.

In this paper I shall deal with the very important question of Cholera convalescents and contacts.

* Scientific Memoirs, Government of India, No. 32.

Methods.

In examining bacteriologically the stools of convalescents and contacts the following technique was adopted.

A considerable portion of the stool was placed in a flask containing peptone water and the flasks were placed in the incubator for 6 hours. At the end of this period a small quantity of the peptone water was taken up from near the surface and a second flask of petone water was inoculated, this was placed in the incubator at 37°C. for about 12 hours: at this point I may observe that Crendiropoulo[a] in his examination of Cholera stools recommended also at least 12 hours in peptone water. A sample from the surface of this peptone water was plated on Dieudonné medium and ordinary Agar by means of an L. shaped spreader as described by me in my research on Enteric fever.[b] The plates were incubated for about 18 hours at 37°C. They were then examined, the same technique was adopted as in the Enteric fever research,[c] namely, all suspicious colonies were picked up and placed in a drop of high titre Cholera agglutinating serum (1-10,000) using a dilution of 1-50 for the purpose. I used a serum prepared by myself by injecting intravenously increasing doses of the living cholera organisms into a rabbit, the cholera vibrio used for this purpose was isolated from a fatal case of cholera. I employed, also, a high titre cholera serum (1-10,000) obtained from the Swiss Serum and Vaccine Institute, Berne, prepared under the supervision of Professor Kolle. Reacting colonies were subcultured and their microscopic and cultural characters studied. Thus each strain was tested microscopically, culturally, and biologically (serum sedimentation).

After these observations were completed I was interested to note that the Scientific Commission of the International Sanitary Conference, Paris, 1911-12, recommended a procedure for the bacteriological examination of Cholera stools on precisely the same lines as stated above.[d]

Also in the case of the prisoners I took a sample of blood from each case in a Wright's capsule; and after the serum had separated I tested it with a typical strain of the Cholera vibrio, and, also, in the case of the "carriers," with their own strain. Various dilutions were put up and the action of the serum was noted both microscopically and macroscopically in the usual manner.

Castor oil was given to some of the convalescents and contacts before the examination to determine whether or not the recovery of the Cholera vibrio from the stool was facilitated thereby.

In the case of some of the convalescents daily examinations of the stools were made for a long period; this is important for the determination of "carriers," since as in the case of enteric fever, the discharge of the organisms in the stools was found to be intermittent. This circumstance considerably increases the difficulties of determining when a person is free from cholera infection.

Examination of Cholera Convalescents.

In the first place I was desirous of knowing the exact number of persons who were discharged from the Cholera Hospital in an infective condition, that is whilst excreting the Cholera vibrio in the stools. For this purpose I examined bacteriologically a number of cases which were being treated and discharged in the usual manner according to the routine of the hospital. I examined in this way the stools of 30 patients and found that 11 of them were still excreting the Cholera vibrio at the time of discharge from hospital and were, therefore, highly infective.

(*a*) Report Sur L' examen des Selles des Voyageurs provenant des pays infectes de Cholera, 1912, Conseil Sanitaire, Maritime et Quarantenaired, Egypt.

(*b*) Report on the methods employed in the campaign against Typhoid fever in Germany. Greig, Jour. R. A. M. C. Volume VI. February 1906.

(*c*) *Ibid* Greig.

(*d*) Bull. de L'offic. intern. d'Hyg. publ. Bd. 4. 1912, No. 2.

The following table shows the name, age, sex, caste of patients, the date of admission to hospital, the date of discharge from hospital, the dates of bacteriological examination of the stools and the presence or absence of the Cholera vibrio in the stools :—

No.	Name.	Age.	Sex.	Caste.	Date of admission.	Date of discharge.	Date of examination of stool.	Character of stool.	Presence or absence of cholera vibrio in stool.
1	Shamacharan ...	32	M.	H.	20th July 1912 ...	23rd July 1912 ...	22nd July 1912 ...	Liquid ...	—
2	Mainabewa ...	55	F.	H.	Do.	25th July 1912 ...	24th July 1912 ...	Liquid ...	+
3	Lakmidas ...	40	M.	H.	18th July 1912 ...	2nd August 1912 ...	Do. ...	Semifluid ...	—
4	Anchi ...	12	M.	H.	19th July 1912 ...	25th July 1912 ...	25th July 1912 ...	Liquid ...	—
5	Mahadaya ...	45	F.	H.	23rd July 1912 ...	27th July 1912 ...	26th July 1912 ...	Do. ...	—
6	Rakhaldas ...	40	M.	H.	26th July 1912 ...	3rd August 1912 ...	27th July 1912 ...	Do. ...	—
7	Tarabewa ...	42	F.	H.	26th July 1912 ...	29th July 1912 ...	28th July 1912 ...	Do. ...	+
8	B. Dass ...	28	M.	H.	23rd July 1912 ...	30th July 1912 ...	29th July 1912 ...	Do. ...	—
9	Bibaran Das ...	25	M.	H.	25th July 1912 ...	Do. ...	Do. ...	Do. ...	—
10	Bhupa Singh ...	25	M.	H.	18th July 1912 ...	4th August 1912 ...	Do. ... 1st August 1912 ...	Do. ... Fluid ...	— +
11	Laksmi ...	45	M.	H.	18th July 1912 ...	2nd August 1912 ...	29th July 1912 ...	Do. ...	+
12	Iswari ...	12	M.	H.	... ...	4th August 1912 ...	Do. ... 4th August 1912 ...	Do. ... Do. ...	+ +
13	Wari Bi ...	38	F.	H.	28th July 1912 ...	2nd August 1912 ...	31st July 1912 ... 1st August 1912 ...	Do. ... Do. ...	— —
14	Thakur Das ...	40	M.	H.	28th July 1912 ...	1st August 1912 ...	31st July 1912 ... 1st August 1912 ...	Do. ... Do. ...	— —
15	Basudeva ...	16	M.	H.	29th July 1912 ...	4th August 1912 ...	31st July 1912 ... 3rd August 1912 ...	Liquid ... Do. ...	— —
16	Gopal Panda ...	35	M.	H.	29th July 1912 ...	1st August 1912 ...	31st July 1912 ...	Fluid ...	—
17	Sungani ...	40	M.	H.	27th July 1912 ...	Do. ...	Do. ...	Soft ...	—
18	Sibram ...	32	M.	H.	25th July 1912 ..	4th August 1912 ...	31st July 1912 ... 1st August 1912 ...	Do. ... Fluid ...	— —
19	B. K. Mukerji ...	36	M.	H.	30th July 1912 ...	1st August 1912 ...	1st August 1912 ...	Do. ...	—
20	Kurpa ...	32	M.	H.	29th July 1912 ...	6th August 1912 ...	2nd August 1912 ... 5th August 1912 ...	Do. ... Do. ...	+ +
21	Bramanando ...	32	M.	H.	Do. ...	4th August 1912 ...	2nd August 1912 ... 4th August 1912 ...	Do. ... Do. ...	+ +
22	Rameswar ...	25	M.	H.	19th July 1912 ...	8th August 1912 ...	3rd August 1912 ... 6th August 1912 ...	Soft ... Do. ...	+ +
23	Jumma Dass ...	45	M.	H.	2nd August 1912 ...	6th August 1912 ...	4th August 1912 ... 5th August 1912 ...	Fluid ... Do. ...	— —
24	Sudarsan ...	39	M.	H.	Do. ...	5th August 1912 ...	4th August 1912 ... 5th August 1912 ...	Do. ... Do. ...	+ +
25	Ruki ...	35	F.	H.	19th July 1912 ...	6th August 1912 ...	5th August 1912 ...	Soft ...	—
26	Sukhali ...	60	F.	H.	20th July 1912 ...	6th August 1912 ...	5th August 1912 ...	Fluid ...	+
27	Janki ...	60	F.	H.	22nd July 1912 ...	3rd August 1912 ...	3rd August 1912 ...	Soft ...	+
28	Narain ...	50	M.	H.	11th August 1912 ...	18th August 1912 ...	18th August 1912 ...	Fluid ...	—
29	Rajani ...	42	M.	H.	12th August 1912 ...	Do. ...	Do. ...	Do. ...	—
30	Chinta ...	50	M.	H.	15th August 1912 ...	Do. ...	Do. ...	Do. ...	—

From this it will be seen that 3·6 per cent. of the cases were discharged from the Cholera Hospital in an infective condition; it is impossible from an ordinary medical examination to say whether or not a patient is infective; it requires special facilities and specially trained observers.

The full significance of this observation will be appreciated when we remember that these pilgrims after discharge from hospital return by train at once to their homes, which are scattered all over India.

The following table shows the various places in India from which pilgrims treated in the Cholera Hospital, Puri, came and to which they returned on discharge and in each case the numbers treated for cholera during July and August 1912:—

Place.	Number.	Place.	Number.
Cuttack	38	Brindaban	4
Rai Bareilly	4	Puri	94
Bharatpur (U. P.) ...	2	Ajoygar	4
Cawnpur	8	Purulia	2
Gaya	5	Hardoi	3
Faridpur	1	Usnatbag	1
Rima	19	Khalikot (Madras) ...	3
Indore	3	Gazipur	1
Punjab	1	Mungee	1
Jaunpur	1	Ranchi	10
Monghyr	3	Ballia	2
Gwalior	18	Joypur (C. P.)	5
Allahabad	3	Sahabad	1
Sitapur	3	Midnapur	23
Rewah	9	Fatepur	3
Muzaffarpur	5	Nababganj	5
Ajodhya (Oudh) ...	3	Bankura	1
Agra	4	Arrah	4
Damoh	2	Gorakhpur	3
Lucknow	1	Bidpur	1
Murshidabad	5	Howrah	3
Sambalpur	2	Hugli	4
Bhilsa	2	Durbhanga	4
Gonda	5	Jhapal	1
Mirzapur	4	Aeedny	2
Bhagalpur	3	Pachora	1
Jubbalpur	7	Athmalik State	1 (Orissa).

Place.	Number.	Place.	Number.
Madura	3	Calcutta	4
Faizabad	1	Hindoi	2
Aligunj	1	Panipat	1
Madras	1	Seetapur	1
Chapra	2	Pryag	2
Jhansi	3	Chandannagar	1
Banda	2	Fusunga	1
Birbhoom	6	Mahirajganj	2
Charkhai	1	Sirai	1
Jhelum	1	Patna	2
Tipparah	1	Basti	1
Champaran	1	Mymensingh	2
Sagar	3	Husunga	1
Tonk	1	Jessore	1
Kustea	1	Balasore	2
Diryambo	2	Sulang	1
Rampur State	1	Narsingpur (U. P.) ...	1
Sylhet	1	Berampore	5
Bezwada (Madras) ...	2	Nipania (U. P.) ...	2
Gurdaspur	1 (U. P.) ...	Bhopal	4
Jhansi	2	Ahmednagar	1
Dhenkanal	1 (Orissa) ...	Delhi	2
Husingabad	1	Rangpur	2
Rajputana	2	Nassik	2
Aligarh	2	Chitrakol	2
Bhairaj	3	Benares	3
Khandapara	3 (Feudatory State).	Silana	1
Khendwa	1	Narsingpur State ...	2 (Feudatory).
Kujur	1	Pratapgarh	2
Ajmir	1	Ganjam	3
Nayagarh State	4 (Feudatory)	Bankipur	1
Surat	1		

A study of this table shows the very wide distribution of the points in India to which these convalescent pilgrims go after discharge from hospital; and, consequently, the dissemination of the Cholera virus throughout the length and breadth of India is taking place, because as we see a number of the Cholera convalsecents harbour the Cholera vibrio and are still infective at the time they leave Hospital and embark in the train for their homes, which they reach in a few days at most.

In order to give a more vivid impression of the distribution of the points in India to which Cholera convalescents go from Puri, I have constructed a sketch map which shows some of the more important places to which Cholera convalescents from the Hospital at Puri went.

To give some idea of enormous numbers of persons moving to and from Puri during the Car Festival, I give the figures showing the arrivals and departures by train day by day during the month of July 1912.

In addition to the pilgrims who came by train a large number also poured into Puri by road. The population of Puri is roughly 50,000. It is estimated that at least 300,000 pilgrims were present on the day of the Car pulling.

The following table shows the number of pilgrims who arrived at and departed from Puri by train during the festival of Nutan Kalebar in the month of July 1912 :—

Date.	Arrival.	Weekly total.	Departure.	Weekly total.	Remarks.
1st	938	...	535	...	
2nd	1,136	...	493	...	
3rd	951	...	392	...	
4th	1,120	...	530	...	
5th	1,005	...	482	...	
6th	1,184	...	425	...	Arrival of special trains.
7th	1,508	7,842	502	3,359	
8th	2,325	...	369	...	Two.
9th	3,075	...	558	...	Three.
10th	4,770	...	408	...	Four.
11th	6,419	...	405	...	Four.
12th	10,804	...	449	...	Nine.
13th	15,875	...	483	...	Eleven.
14th	16,946	60,214	350	3,022	Nine.
15th	24,663	...	300	...	Eleven.
16th	20,854	...	10,240	...	
17th	14,896	...	17,675	...	
18th	3,181	...	15,140	...	
19th	2,108	...	14,295	...	
20th	2,183	...	13,965	...	
21st	2,285	70,170	11,627	83,242	
22nd	4,689	...	12,585	...	
23rd	3,156	...	2,212	...	
24th	3,915	...	5,548	...	
25th	1,216	...	5,520	...	
26th	1,234	...	4,896	...	
27th	1,014	...	4,071	...	
28th	1,042	16,311	2,561	37,391	
29th	1,540	...	2,427	...	
30th	1,542	...	2,910	...	
31st	1,530	4,612	1,347	6,684	
Grand total	...	159,149	...	133,698	

In the next place I endeavoured to determine the duration of the infectivity of persons recently recovered from Cholera, and for this purpose I was fortunate enough to have the opportunity of studying an epidemic in a jail, and, consequently, I was able to keep up my daily bacteriological observations for a prolonged period which I could not have done amongst the free population. A Table in the appendix to this paper gives the name, age, caste, sex of prisoners, date of attack by cholera, the dates of examination, the body weight, the presence or absence of cholera agglutinins in the blood, and the presence or absence of the Cholera vibrio in the stools.

Examination of Cholera Contacts.

I examined a number of healthy men in contact with Cholera cases in hospital. As these persons were attached to the Cholera Hospital as temporary staff they formed more suitable material for the observations than a constantly moving population here to-day and gone to-morrow.

I examined 27 persons presenting no signs of disease and found 6 persons excreting the Cholera vibrio in the stools.

The following table shows the name, age, sex, caste, occupation of contacts, date of examination, the character of stool, and the presence or absence of the Cholera vibrio in the stool :—

No.	Name.	Age.	Sex.	Caste.	Occupation.	Date of examination of stool.	Character of stool.	Presence or absence of cholera vibrio in stool.
1	Anti ...	25	M		Sweeper ...	23rd July 1912 ...	Formed ...	—
						27th July 1912 ...	Do. ...	—
						3rd August 1912 ...	Do. ...	—
						7th August 1912 ...	Do. ...	—
2	Madna ...	23	M	H	Ward attendant.	23rd July 1912 ...	Do. ...	—
						25th July 1912 ...	Do. ...	+
						29th July 1912 ...	Do. ...	+
3	Bidi ...	35	M	H	Sweeper ...	24th July 1912 ...	Do. ...	—
4	Sanninaik ...	40	M	H	Do. ...	24th July 1912 ...	Do. ...	+
						25th July 1912 ...	Do. ...	+
						28th July 1912 ...	Do. ...	+
						2nd August 1912 ...	Do. ...	+
						3rd August 1912 ...	Do. ...	—
5	Shoday ...	29	M	H	Do. ...	24th July 1912 ...	Do. ...	—
						28th July 1912 ...	Do. ...	—
6	Shanto ...	23	M	H	Do. ...	24th July 1912 ...	Do. ...	—
						29th July 1912 ...	Do. ...	—
7	Nuchi* ...	40	M	H	Do. ...	25th July 1912 ...	Do. ...	+
						26th July 1912 ...	Do. ...	+
						27th July 1912 ...	Do. ...	+
						2nd August 1912 ...	Do. ...	+
8	Rajan ...		M	H	Do. ...	25th July 1912 ...	Do. ...	—
9	Madna ...	35	M	H	Do. ...	25th July 1912 ...	Do. ...	—
						2nd August 1912 ...	Do. ...	—
						7th August 1912 ...	Do. ...	—
10	Arjun ...	40	M	H	Do. ...	25th July 1912 ...	Do. ...	—
11	Felao ...	30	M	H	Do. ...	25th July 1912 ...	Do. ...	—
12	Pillai ...	40	F	H	Do. ...	26th July 1912 ...	Do. ...	—

* He appears quite healthy; he left his employment at the Hospital on August 1st. It was ascertained that he was sick with "fever" and "dysentery" from 2nd to 21st August. He returned to duty in the Municipality on August 22nd.

No.	Name.	Age.	Sex.	Caste.	Occupation.	Date of examination of stool.	Character of stool.	Presence or absence of Cholera vibrio in stool.
13	Rahchi ...	50	F	H	Sweeper ...	26th July 1912 ...	Formed ...	—
14	Chuni ...	40	F	H	Do. ..	Do. ...	Do. ...	—
15	Magia ...	35	F	H	Do. ...	Do. ...	Do. ...	—
16	Wolimini ...	42	F	H	Do. ...	Do. ...	Do. ...	—
17	Sreemati ...	50	F	H	Do. ...	Do. ...	Do. ...	—
18	Sonai ...	30	M	H	Do. ...	27th July 1912 ...	Soft ...	—
						7th August 1912 ...	Do. ...	—
19	Manna ...	30	M	H	Do. ...	28th July 1912 ...	Formed ..	—
						29th July 1912 ...	Do. ...	—
20	Bidya ...	28	M	H	Do. ...	27th July 1912 ...	Do. ...	—
						28th July 1912 ...	Do. ...	—
21	Fatra —	30	M	H	Do. ...	29th July 1912 ...	Soft ...	—
						2nd August 1912 ...	Do. ...	—
						3rd August 1912 ...	Do. ...	+
						7th August 1912 ...	Do. ...	+
22	Bidamag ...	35	M	H	Do. ...	29th July 1912 ...	Formed ...	—
23	Chandra (*a*) ...	40	M	H	Ward attendant	20th July 1912 ...	Do. ...	+
24	Bhutto ...	30	M	H	Sweeper ...	3rd August 1912 ...	Do. ...	—
25	Hurry ...	40	M	H	Do. ...	7th August 1912 ...	Do. ...	—
26	Kandle naik ...	38	M	H	Do. ...	7th August 1912 ...	Do. ...	—
27	Bhubu naik ...	30	M	H	Do. ...	7th August 1912 ...	Do. ...	+

In considering these observations it will be convenient to discuss each group.

(*1*) *Cholera cases discharged from hospital while still infective.*—From a study of the results of the investigation of the cases it will be seen that the bacteriological examination showed that the Cholera vibrio was being excreted in the stools of about one-third of the cases examined. When it is remembered that these infective Cholera convalescents in most cases left Puri at once by train and, as has been shown, their destinations are scattered widely over India, the gravity of this factor in the propagation of Cholera will be obvious. Further, it has also to be taken into consideration that although a certain number of these cases may cease in a short time to excrete the Cholera vibrio in the stools, "temporary Cholera foci," yet amongst them there are others, "chronic carriers," who will go on excreting intermittently the organism for long periods and act as "permanent foci of infection." As I have shown,(*b*) the comma bacillus in about one-third of the cases of Cholera gains an entrance to the gall-bladder, where it finds all the conditions favourable for its prolonged life. Such individuals are especially dangerous because they are reservoirs of the Cholera virus; and they bear the same relation to Cholera as the big game of Africa do to Nagana. These "carriers" enable the Cholera vibrio to maintain its existence by protecting it and affording it suitable conditions for its life. It is obvious that the detection of these healthy "carriers" is all-important. In discussing the daily observations on convalescent prisoners, I shall return to this point again. In the light of the fact that the comma bacillus can live in the gall-bladder possibly for long periods and is excreted into the external world in the fæces, some unexplained difficulties in the epidemiology of Cholera are cleared up. Further, when we keep before us such a table as the above showing the widely separated points in India to which infected individuals return from the Puri pilgrimage and mix freely with their fellows, the wide incidence of the disease in India is not to be wondered at. These facts show the great, but I trust not insuperable,

(*a*) Became an acute case of Cholera and died 23rd July 1912.

(*b*) An investigation on the occurrence of the Cholera vibrio in the biliary passages.

difficulties which have to be faced in dealing with the problem of the prevention of Cholera.

Devecchi, B and Randone, Fr.(*a*) have found that in 115 Cholera convalescents examined bacteriologically, the duration of the vibrio in the stool varied between a minimum of two and a maximum of 35 days; and in 57 healthy contacts the duration varied between a minimum of two and a maximum of 13 days.

The International Sanitary Conference at Paris (1911) report(*b*) that the duration of the excretion of the vibrios by the Cholera carrier is as a rule short (2 to 3 weeks) : exceptionally it lasts to 12 months. The excretion is irregular : and there are bacilli free intervals of 8 to 12 days. This is in agreement with my own observations.

2. The daily examination over a prolonged period of the stools of prisoners convalescent from Cholera.—In my research on Enteric fever in India(*c*) I carried out a number of daily bacteriological observations on Enteric convalescents and found that a certain number of them continued to exerete the B typhosus intermittently for prolonged periods. In the case of Cholera I find that although in the majority of cases the excretion appears to cease very shortly after the acute attack, yet in 3 cases out of 11 examined daily for a considerable period the Cholera vibrio was found at intervals in the stools for longer periods. As I have shown,(*d*) infection of the bile by the comma bacillus occurs in about one-third of the Cholera cases, a lower percentage than in Enteric, and, probably, a lower percentage of "chronic carriers" will be found in Cholera than in Enteric fever, but the enormous number of cases of Cholera in India has to be taken into account, and further as therapeutic methods improve and a higher percentage of cures is attained, the number of "carriers" will increase. The reduction of mortality is undoubtedly to be aimed at, but, from the point of view of the community at large, having regard to the increased production of Cholera "carriers" produced thereby it is not an unmixed blessing, at least in the present state of our knowledge of methods of prevention of Cholera. In cases Nos. 3 and 26, appendix, in which the prolonged excretion of the Cholera vibrio was noted there were long intervals, as in Enteric fever, in which the stools remained free from Cholera vibrios. Castor oil was given on several occasions but did not appear to have any effect on the excretion of the comma bacillus. An interesting point was the detection on several occasions in the stools, both of the "carriers" and the "non-carriers," especially when these were loose, of other vibrios, these, however, did not react with a high titre agglutinating serum ; such forms might easily mislead an inexperienced worker and, indeed, before the biological test was introduced some of these forms might have been mistaken for the Cholera vibrio by experienced observers. It is interesting to note that the International Sanitary Conference, Paris, 1911 (*e*), refer to this difficulty. Bernhardt, George(*f*) also deals with this point. Crendiropoulo(*g*) is disposed to regard the finding of non-agglutinating vibrios in the stools of persons coming from areas where Cholera is epidemic as sufficient evidence to place the "carrier" of such organisms under suspicion. This view, however, requires confirmation.

Examination of the blood of Cholera convalescents.—I tested the blood of all Cholera convalescent prisoners. I obtained very interesting results, namely, the two prisoners, Cases No. 3 and 26, Appendix, who continued to harbour the Cholera vibrio showed a definite reaction with the sedimentation test against a standard strain of Cholera vibrio, whilst all the others who did not show the comma bacillus in their stools gave a negative reaction. Further I tested one of the prisoners (carrier), Case No. 26. Appendix, using his own strain

(*a*) Alcane Osservazioni betteriologiche e. statische praticate durante l'epidemia Colerica nella provincia di Siracusa. (Pathologica. 1912. No. 87 page 347.)

(*b*) Bull. de l'offic. intern. d. Hyg. publ. Bd. 4. 1912. No. 2.

(*c*) Scientific Memoir of the Government of India No. 32.

(*d*) *Ibid.*

(*e*) *Ibid.*

(*f*) Zeitschr. f. Hyg. u. Infectionskrankheiten. Bd. 71 H, 3. S. 435. 1912.

(*g*) *Ibid.*

of the Cholera organism and his own serum and found that a distinct reaction was given.

Massaglia (*a*) also found agglutinins in the blood of Cholera carriers. Deboni F. (*b*) did not find any agglutinins or bacteriolysines in the blood of contacts(carriers).

This is a somewhat important observation as the Widal test may be used as a means of detecting possible "carriers" amongst a number of individuals, and in the case of a positive reaction being obtained a bacteriological examination of the stools should be carried out to determine whether or not the person is excreting the Cholera vibrio.

That convalescent "carriers" can produce epidemics of Cholera was demonstrated by the outbreak in the Jail which I deal with in another paper. (*c*)

(3) *Cholera contacts.*—In our researches on Enteric fever in India (*d*) we dealt with the question of enteric nursing orderlies and we showed that some of these men contracted the disease and died of it, but others, and they are the most important from the preventive point of view, became infected with the B typhosus and showed no signs of ill health, they continued to excrete B typhosus in their stools, and on returning to their barracks formed fresh foci of infection. In cholera a very similar position presents itself. Some of the attendants on cholera cases contract the disease and die of it, whilst, on the other hand, some become infected but remain apparently quite healthy. On examining the stools of healthy persons I found that out of 27 bacteriologically investigated, 6 harboured the cholera vibrio and excreted it in their stools. Obviously such persons are particularly dangerous, because equally infective with the severe case, yet, whilst in the latter the clinical signs at once attract attention and are, indeed, danger signals, the former pass undetected and their dejecta remain undisinfected, so the contacts form an important factor in the dissemination of the disease, and one that has to be reckoned with in any scheme for preventing or limiting epidemics.

The International Sanitary Conference, Paris, 1911, report (*e*) that in Rotterdam and Amsterdam 7,338 travellers from suspected ports in the Baltic were examined and 7 Cholera carriers found ; at Naples 2,000 emigrants were examined and 12 Cholera carriers discovered. In Egypt 15,000 persons from suspected ports were examined and 22 vibrio carriers detected. At St. Petersburg, on the contrary among 9,737 persons, in contact with cholera patients 377 vibrio carriers were found. In Austria the number of carriers was 50 % of the sick.

Anderson(*f*) states that in New York about 34,000 stools of travellers by sea were examined and in 56 Cholera vibrios were determined : in 28 cases the persons were actually sick, in 27 cases they were healthy vibrio carriers.

The question of dealing with the Cholera convalescents and contacts.—In the present state of our knowledge this problem presents very considerable practical difficulties. An ideal method, which is to be hoped may be reached in time and which would greatly help in the eradication of the disease, would be by Chemo-therapy ; if a specific drug were available for the destruction of the comma bacillus in the tissues of the host without damaging the latter, *i.e.*, disinfection of the host as regards cholera, then a very strong weapon would be in our hands for the prevention of cholera. Meantime we have to fall back on—

(1) *Disinfection of the comma bacilli outside the host.*—and the essential points to bear in mind are, that disinfection of fresh night soil should be carried out as early and as extensively as possible. I have considered this question in another paper(*g*). From practical experience I have found that chlorinated

(*a*) Soc med-chir. di Modena. Sitz des 10-2-1.

(*b*) Richerche sui portatori sani di vibrioni colerigeni (Pathologica. 1912, No, 87 page 341).

(*c*) Epidemic of cholera in a Jail caused by a carrier. Major E. D. W. Greig.

(*d*) Scientific Memoir of Government of India, No. 32.

(*e*) Bull de l'offic. intern. d'Hyg. publ. Bd. 4. 1912 No. 2.

(*f*) Jour. Amer. Med. Assoc. Vol. 58, 1912, No. 23, page 1728.

(*g*) Disinfection in Cholera. Major E. D. W. Greig, I. M. S.

lime (32 per cent. chlorine) has many advantages and is very efficacious for the disinfection of fresh night soil(*a*).

(2) *The segregation and examination of convalescents and contacts.*—As I have shown in my paper on Enteric fever in India(*b*) the method of isolation and bacteriological examination of enteric convalescents in the British Army in India has been eminently successful. In regard to such an application to the general population in India in the case of cholera, there would be great practical difficulties. At the same time the question is worthy of careful consideration and a suitable modification might be arrived at.

In dealing with this problem our forces might be concentrated at the most important points; and there can be little doubt that the great pilgrim centres are, as regards the propagation of cholera in India, the greatest danger points, and should receive particular attention. A longer period of segregation of the cholera patient at these centres should be arranged for, and coupled with this observations should be made by trained bacteriological workers. At such centres many valuable facts would accumulate which would guide us in dealing with the prevention of Cholera in the future.

The recent work of Crendiropoulo(*c*) is of interest in this relation as showing the large number of stools which were examined for cholera. He has examined the stools of 34,461 persons arriving by ship at Alexandria from Cholera infected ports. He found only 23 true "Cholera carriers," and 40 persons with non-agglutinating vibrios in their stools. The carriers are found chiefly amongst the passengers, seldom amongst the crew of the vessels: Crendiropoulo regards a "carrier" as free from infection if the stools are negative after 3 or 4 examinations carried out at intervals of 3 or 4 days. As I have shown in this paper the excretion of the cholera vibrio in the fæces of the "carrier" is markedly intermittent and there may be long "bacilli free" periods; *e.g.*, in case 3, 21 days. Hence the above test would be inadequate in such cases.

(*a*) Disinfection in Cholera, Major E. D. W. Greig, I.M.S.

(*b*) Enteric fever in India—An account to date of the results obtained from the practical working of the recommendations of the Enteric Research Committee of the Government of India (1906-08), Major E. D. W. Greig.

(*c*) *Ibid.*

APPENDIX.

Table showing the result of serum sedimentation test (Widal reaction), the presence or absence daily of the Cholera vibrios in the stools, the character of the stool, and the body weight of the prisoner.

This is an important observation as it shows the results of a daily bacteriological examination of stools of persons convalescent from Cholera and carried out for a prolonged period. This investigation was made possible by the patients being prisoners; in a free population it would have been practically impossible. So far as I am aware daily observations on convalescent cholera patients for a prolonged period have not been carried out hitherto.

The attention of those engaged in the prevention of Cholera is directed to this table.

13

DAILY OBSERVATIONS

ON

CONVALESCENT PRISONER.

No. 1.

Name—Iswari, Hindu, Male.

Age—12.

Date of attack—July 6th 1912.

Discharged from Cholera Hospital—July 13th 1912.

Admitted to Jail—July 23rd 1912.

Date of examination of stools.	Widal reaction Cholera vibrio.	Presence or absence of Cholera vibrios in stools.	Character of stools.	Body weight in lbs.
July 6th	...	Acute attack of Cholera.	...	...
„ 7th	...	...	...	...
„ 8th	...	...	...	...
„ 9th	...	...	...	...
„ 10th	...	...	...	...
„ 11th	...	...	...	...
„ 12th	...	...	...	...
„ 13th	...	...	...	...
„ 23rd	...	...	...	...
„ 28th	...	+ + +	Formed.	...
„ 30th	...	+ + +	„	...
August 1st	...	+ + +	„	...
„ 3rd	...	+ + +	„	...
„ 4th	...	+ + +	„	...

REMARKS.—This is a very interesting and important case. This "carrier" caused an outbreak of Cholera in the Jail, 17 cases and 5 deaths. He was attacked on July 6th, discharged from Hospital, July 13th, arrested and sent to Jail on 23rd, and cases began to occur in the Jail a few days after his admission and in the same section. He was excreting the cholera vibrios in large numbers in his stool at the time of entrance to Jail and did so until he was released, August 4th, that is for nearly a month. He returned by train to his home near Agra being still infective.

DAILY OBSERVATIONS

ON

CONVALESCENT PRISONER.

No. 2.

Name—Chowdhury Naik, Hindu, Male.

Age—35.

Date of attack—27th July 1912.

Date of examination of stools.	Widal reaction Cholera vibrio.	Presence or absence of Cholera vibrios in stools.	Character of stools.	Body weight in lbs.
July 21st	...	...	...	109
„ 27th	...	+	...	...
„ 29th	...	—	...	...
August 1st	...	—	...	...
„ 2nd	...	—	...	...
„ 3rd	...	—	...	...
„ 10th	...	—	...	...
„ 11th	...	—	...	...
„ 12th	...	—	...	...
„ 18th	...	—	...	95
„ 19th	...	—	...	...
„ 20th	...	—	...	...
„ 21st	...	—	...	...
„ 23rd	...	—	...	...
„ 25th	...	—	...	88
„ 27th	...	—	...	...
„ 29th	...	—	...	...
„ 31st	...	—	...	...
September 1st	...	—	...	92
„ 2nd	...	—	...	...
„ 3rd	...	—	...	...
„ 4th	...	—	...	...
„ 5th	...	—	...	...
„ 6th	...	—	...	...
„ 7th	10 30 60 100	—	...	Ol. Ricin Ziv
„ 8th	...	—	...	94
„ 9th	...	—	...	...
„ 10th	...	—	...	...
„ 11th	...	—	...	...
„ 12th	...	—	...	...
„ 13th	...	(Non-Agglutinating vibrios present.)	...	...
„ 14th	...	—	...	...
„ 15th	...	—	...	95

REMARKS.—After the acute attack cholera vibrios were not detected in the stools, although on one occasion non-agglutinating vibrios were found. The blood contains no cholera agglutinins.

15

DAILY OBSERVATIONS

ON

CONVALESCENT PRISONER.

No. 3.

Name—Babaji Mahapatra, Hindu, Male.

Age 27.

Date of attack, 31st July 1912.

Date of examination of stools.	Widal reaction Cholera vibrio.	Presence or absence of Cholera vibrios in stools.	Character of stools.	Body weight in lbs.
July 21st	...	—	...	110
August 1st	...	+	...	...
,, 3rd	...	+	...	=
,, 4th	...	—	...	...
,, 5th	...	—	...	...
,, 6th	...	—	...	...
,, 11th	...	—	...	...
,, 12th	...	—	...	...
,, 18th	...	—	...	95
,, 19th	...	—	...	...
,, 20th	...	—	...	...
,, 21st	...	—	...	...
,, 22nd	...	—	Fluid stool.	...
,, 23rd	...	—	Ditto	...
,, 24th	...	+ +	Soft with mucous.	...
,, 25th	...	+ +	Ditto	90
,, 26th	...	—	Ditto	...
,, 27th	...	—	Semifluid without mucous.	...
,, 28th	...	—	...	...
,, 29th	...	—	...	...
,, 30th	...	—	...	...
,, 31st	...	—	Ol. Ricini Ziv	93
September 1st	...	—	Ol. Ricini Ziv	93
,, 2nd	...	(Non-Agglutinating vibris).	...	...
,, 3rd	...	—	...	...
,, 4th	...	—	...	...
,, 6th	...	—	...	...
,, 7th	+ + ± — 10 30 60 100	—	...	...
,, 8th	...	—	...	98
,, 9th	...	—	...	...
,, 10th	...	—	...	...

No. 3—*concld.*

Daily examination of stools.	Widal reaction Cholera vibrio.	Presence or absence of Cholera vibrios in stools.	Character of stools.	Body weight in lbs.
September 11th	..	—	...	...
„ 12th	+ + ± — — 10 30 60 90 120	+ +	...	...
„ 13th	...	+ +	...	...
„ 14th	...	—	....	...
„ 15th	+ + + + ± 10 20 30 50 60	—	...	98
„ 16th	(With autogenous strain Cholera vibrio.)	—	...	...
„ 17th	...	—	...	...
„ 18th	+ + + + + 20 40 60 80 100	—	...	...
„ 19th	...	—	...	...
„ 20th	...	—	...	...

REMARKS.—This is an important case. The Cholera Vibrios were recovered from his stools 6 weeks after the attack. The discharge of the vibrios was markedly intermittent as in the case of B. typhosus. It is interesting to note that Cholera like vibrios occurred in the stool of this man on one occasion but they did not react with a high titre agglutinating serum. His blood also contained Cholera agglutinins.

17

DAILY OBSERVATIONS
ON
CONVALESCENT PRISONER.

No. 6.
Name—Binode Bhui, Hindu, Male.
Age 23.
Date of attack—22nd July 1912.

Date of examination of stools.	Widal reaction Cholera vibrio.	Presence or absence of Cholera vibrios in stools.	Character of stools.	Body weight in lbs.
July, 21st				96
August, 2nd		+ +		
,, 3rd		+ +		
,, 4th		+		
,, 5th		+		
,, 8th		—		
,, 9th		—		
,, 12th		—		
,, 18th		—		97
,, 19th		—		
,, 20th		—		
,, 22nd		—		
,, 23rd		—		
,, 24th		—		
,, 25th		—		
,, 26th		—		
,, 27th		—		
,, 28th		—		
,, 29th		—		
,, 30th		—	Ol. Ricini Ziv	
,, 31st		—		
September, 1st		—		93
,, 2nd		—		
,, 3rd		—		
,, 4th		—		
,, 5th		—		
,, 6th		—		
,, 7th		—		
,, 8th		—		
,, 9th		—		
,, 10th		—		
,, 11th	10 30 50 100	—		
,, 12th		—		
,, 13th		(Non-agglutinating vibrios.)		
,, 14th		—		
,, 15th		—		98

REMARKS.—The 3rd day after the attack was the last occasion on which cholera vibrios were found in this patient's stools although he has been examined daily since.

18

DAILY OBSERVATIONS

ON

CONVALESCENT PRISONER.

No. 9.

Name—Ajij Khan.

Age 23.

Date of attack—5th August 1912.

Date of examination of stools.	Widal reaction Cholera vibrio.	Presence or absence of Cholera vibrios in stools.	Character of stools.	Body weight in lbs.
July, 21st				84
August, 6th		+		
" 7th		+		
" 8th		—		
" 10th		—		
" 18th		—		84
" 19th		—		
" 20th		—		
" 21st		—		
" 22nd		—		
" 23rd		—		
" 24th		—		
" 25th		—		
" 26th		—		
" 28th		—		
" 29th		—	Ol. Ricini Ziv	
" 30th		—	Soft with mucous.	
September, 1st		—	Ol. Ricini Ziv	83
" 2nd		—		
" 3rd		—		
" 4th		—		
" 5th		—		
" 6th		—		
" 7th		—		
" 8th		—		
" 9th		—		
" 10th	10 20 50 100	—		
" 11th		—		
" 12th		—		
" 13th		—		
" 14th		—		
" 15th		—		84

REMARKS.—This case did not continue to excrete the comma bacillus after the acute attack.

19

DAILY OBSERVATIONS
ON
CONVALESCENT PRISONER.

No. 10.
Name—Durlav Naik, Hindu, Male.
Age 19.
Date of attack—7th August 1912.

Date of examination of stools.	Widal reaction Cholera vibrio.	Presence or absence of Cholera vibrios in stools.	Character of stools.	Body weight in lbs.
July, 21st,...	...	...	...	97
August, 8th	...	+	...	
,, 9th	...	+	...	
,, 10th	...	+	...	
,, 11th	...	—	...	
,, 12th	...	—	...	
,, 18th	...	—	...	86
,, 19th	...	—	...	
,, 21st	...	—	...	
,, 22nd	...	Not examined.	...	
,, 23rd	...	—	...	
,, 24th	...	—	...	
,, 25th	...	—	...	90
,, 26th	...	—	...	
,, 28th	...	—	...	
,, 29th	...	—	...	
,, 31st	...	—	Ol. Ricini Ziv	
September, 1st	...	—	...	90
,, 2nd	...	—	...	
,, 3rd	...	(Non-agglutinatine vibrios.)	...	
,, 4th	...	—	...	
,, 5th	...	—	...	
,, 6th	...	—	...	
,, 7th	...	—	...	
,, 8th	...	—	...	90
,, 9th	...	—	...	
,, 10th	...	—	...	
,, 11th	10, 30, 50, 100	—	...	
,, 12th	...	—	...	
,, 13th	...	(Non-agglutinating vibrios.)	...	
,, 14th	...	—	...	
,, 15th	...	—	...	90

REMARKS.—In this case the excretion of the Cholera vibrio ceased after recovery from the acute attack and the vibrios did not reappear. Vibrios which did not agglutinate with a high titre Cholera serum were met with on two occasions in the stools.

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DAILY OBSERVATIONS

ON

CONVALESCENT PRISONER.

Date

No. 11.

Name—Bidum Swami, Hindu, Male.

Age 19.

Date of attack—8th August 1912.

Date of examination of stools.	Widal reaction Cholera vibrio.	Presence or absence of Cholera vibrios in stools.	Character of stools.	Body weight in lbs.
July, 21st	...	...	...	126
August, 8th	...	+	...	
„ 9th	...	+	...	
„ 10th	...	+	...	
„ 11th	...	+	...	
„ 22nd	...	+	...	
„ 23rd	...	—	...	
„ 24th	...	—	...	
„ 25th	...	—	...	115
„ 26th	...	—	...	
„ 27th	...	—	...	
„ 28th	...	—	...	
„ 30th	...	—	...	
September 1st	...	—	...	110
„ 2nd	...	—	...	
„ 3rd	—	Non-agglutinating vibrio.	...	
„ 4th	...	—	...	
„ 6th	...	—	...	
„ 7th	...	—	...	
„ 8th	...	—	...	114
„ 9th	...	—	...	
„ 10th	...	—	...	
„ 11th	...	—	...	
„ 12th	...	—	...	
„ 13th	‡ — — — 10, 30, 50, 100	(Non-agglutinating vibrio.)	...	
„ 14th	...	—	...	
„ 15th	...	—	...	115
„ 16th	...	—	...	
„ 17th	...	—	...	

REMARKS.—This man continued to excrete the Cholera vibrios for 14 days after the acute attack. Since then they have not reappeared in his stools although daily bacteriological examinations were made. On two occasions after this, September 3rd and 13th, vibrios were recovered from the stools, but they were not agglutinated by a high titre Cholera serum.

DAILY OBSERVATIONS

ON

CONVALESCENT PRISONER.

No. 23.

Name—Brindaban Panda, Hindu, Male.

Age 16.

Date of attack—12th August 1912.

Date of examination of stools.	Presence or absence of Cholera vibrios in stools.	Character of stools.
August, 11th	+	
„ 13th	+	
„ 19th	—	
„ 20th	—	
„ 22nd	—	
„ 23rd	—	
„ 24th	—	
„ 26th	—	
„ 27th	—	
„ 28th	—	Ol. Ricini. Ziv.
„ 29th	—	Soft with mucous.
„ 30th	—	Soft stool.

REMARKS.—Apparently this man ceased to excrete the Cholera vibrio after recovery from the acute attack.

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DAILY OBSERVATIONS

ON

CONVALESCENT PRISONER.

No. 26.

Name—Nanhu, Male,—Hindu.

Age 30.

Date of attack—11th August 1912.

Date of examination of stools.	Widal reaction Cholera vibrio.	Presence or absence of Cholera vibrios in stools.	Character of stools.	Body weight in lbs.
July, 21st	...	...	...	112
August, 12th	...	+	...	
„ 18th	...	—	...	102
„ 19th	...	—	...	
„ 20th	...	—	...	
„ 21st	...	—	...	
„ 23rd	...	—	...	
„ 24th	...	—	...	
„ 25th	...	—	...	97
„ 26th	...	+ +	Dark fluid with mucous.	
„ 27th	...	+ +	Do.	
„ 28th	...	—	Do.	
„ 29th	...	—	Do. Ol. Ricini Ziv.	
„ 30th	...	—	Fluid with mucous.	
„ 31st	...	—	...	
September, 1st...	...	—	...	102
„ 2nd	...	—	...	
„ 3rd	...	— (Non-agglutinating vibrios.)	...	
„ 4th	...	—	...	
„ 5th	...	—	...	
„ 6th	...	—	...	
„ 7th	...	—	...	
„ 8th	...	—	...	102
„ 9th	...	—	...	
„ 10th	...	—	...	
„ 11th	+ + + ‡ 10 30 50 100	— (Non-agglutinating vibrios.)	...	
„ 12th	...	—	...	
„ 13th	+ + ± — — 10 30 60 120 160	—	...	
„ 14th	...	—	...	

No. 26—*concld.*

Name—Nanhu, Male, Hindu.

Age 30.

Date of attack—11th August 1912.

Date of examination of stools.	Widal reaction Cholera vibrio.	Presence or absence of Cholera vibrios in stools.	Character of stools.	Body weight in lbs.
September 15th	...	—	...	112
„ 16th	‡ — — — 10 20 30 60 (with autogenous non-agglutinating vibrio).	—	...	
„ 17th	...	—	...	
„ 18th	...	—	...	
„ 19th	...	—	...	
„ 20th	...	—	...	

REMARKS.—This is an interesting case. This man was attacked by Cholera on the 11th of August and immediately after the acute attack the comma bacillus was not recovered, but after a considerable interval, 16 days, the organism was detected in the stool. It is interesting to note that later on a vibrio was cultivated from the stool, but on testing it with a high titre Cholera serum it did not agglutinate. This illustrates the pitfalls and difficulties in the examination of Cholera "carriers." The blood serum of this case contained Cholera agglutinins, it is probable that he had an infection of the bile. His serum did not agglutinate the vibrio cultivated from his stool which was not acted on by the high titre Cholera serum.

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DAILY OBSERVATIONS
ON
CHOLERA CONTACT PRISONER.

No. 30.
Name—Khettra, Male, Hindu.
Age 30.
Attendent on Cholera patients inside Jail.

Date of examination of stools.	Widal reaction Cholera vibrio.	Presence or absence of Cholera vibrios in stools.	Character of stools.	Body weight in lbs.
July, 21st	...	—	...	116
August, 18th...	...	—	...	103
„ 19th	...	—	...	...
„ 20th	...	—	...	...
„ 21st	...	—	...	...
„ 22nd	...	—	...	...
„ 23rd	...	—	...	...
„ 24th	...	—	...	...
„ 25th	...	—	...	107
„ 26th	...	—	...	...
„ 27th	...	—	...	...
„ 30th	...	—	...	...
„ 31st	...	—	...	...
September, 1st	...	—	Ol. Ricini. Ziv	109
„ 2nd	...	—	...	...
„ 3rd	...	—	...	...
„ 4th	...	—	...	...
„ 5th	...	—	...	...
„ 6th	...	—	Ol. Ricini Ziv	...
„ 7th	...	—	...	...
„ 8th	...	—	...	106
„ 9th	...	—	...	...
„ 10th	...	—	...	...
„ 11th	...	—	...	...
„ 12th	— — — 10 30 50	—	...	...
„ 13th	...	—	...	...
„ 14th	...	—	...	...
„ 15th	...	...	...	115
„ 16th	...	...	...	...
„ 17th	...	...	...	...
„ 18th	...	...	...	...
„ 19th	...	...	...	...
„ 20th	...	...	...	...
„ 21st	...	...	...	...

REMARKS.—This man, a prisoner, was an attendent on a cholera case. Daily examination of his stools was made over a prolonged period in order to detect, if present, an intermittent discharge of the Cholera vibrio. He had not become a "carrier."

ALL-INDIA SANITARY CONFERENCE,—MADRAS, NOVEMBER 1912.

AN INVESTIGATION OF AN EPIDEMIC OF CHOLERA CAUSED BY A "CARRIER"

By

Major E. D. W. Greig, M.D., D.Sc.; I.M.S. On special duty for the Cholera Enquiry.

Owing to its being the celebration of the Nutan Kalebar, the number of pilgrims present during the Car Festival at Puri this year was very great. It was roughly estimated that about 300,000 attended. They came from all over India. The annual cholera epidemic was, consequently, unusually severe ; 417 cases were admitted to hospital in July. After the ceremony in July was over, the town of Puri became severely infected with cholera and the number of cases returned daily reached 49 on the 30th August.

During July and August this year Cholera occurred in the Jail at Puri. I was able to make a very complete study of this epidemic. Before entering into details I may briefly summarise the facts. On investigation it was found that a patient who had been attacked by Cholera on the 6th July 1912 was discharged from the Cholera Hospital, Puri, and was wandering about until his arrest and admission to Jail on the 23rd July 1912. I made a careful bacteriological examination of the stools of this man, who was in the undertrial ward of the Jail, and found that he was excreting the Cholera vibrio in large numbers at the date of examination, 28th July 1912, that is, 3 weeks after date of attack. A few days after his admission to the Jail, cases of Cholera began to occur in the undertrial ward. Including warders, 17 cases of Cholera with 5 deaths took place in the Jail as a result of the introduction of the Cholera virus by this "carrier." The "carrier" was at once segregated, but before this was done he had an opportunity of infecting latrines and flies ; the latter were very numerous in the Jail and elsewhere in Puri at that time. It had also to be remembered that, apart from the men who developed an acute attack of the disease, there were, also, others who, although acquiring the infection, remained healthy or showed very slight signs and would pass undetected. The outbreak was effectively controlled by a very complete disinfection of all fresh night soil in the Jail ; this is considered by me in another communication.*

Methods.—In the examination of the stools of the "carrier," a specimen of the faeces was placed in peptone water and incubated at 37°-C. for 6 hours ; from this a second flask of peptone water was inoculated and incubated about 12 hours at 37°-C. After this enrichment process, a few drops of the peptone water were placed on Dieudonné and ordinary agar plates, which were incubated for 18 hours at 37°-C. At the end of this period the plates were examined and suspected colonies tested with two high titre Cholera sera, one prepared by myself (titre 1-8000), one prepared by the Swiss Serum and Vaccine Institute, Berne, under the supervision of Professor Kolle. Reacting colonies were subcultured and the microscopic and cultural characters carefully studied. In this way the Cholera vibrio separated from the stool was fully tested and authenticated by microscopic, cultural and biological (agglutination) tests.

Bacteriological examination of the "carrier".—The following table gives the date of attack by Cholera, the date of his admission to Jail, the dates of the examination of and the presence or absence of Cholera vibrio in the stools of the "carrier."

*Observations on disinfection in Cholera, Major E. D. W. Grieg, I.M.S.

Name.	Age.	Sex.	Caste.	Date of attack.	Date of admission to Jail.	Date of examination of stools.	Presence or absence of Cholera vibrio in stools.
I	12	M	H	6th July 1912 ...	23rd July 1912 ...	28th July 1912 ...	+ + +
						30th July 1912 ...	+ + +
						1st August 1912 ...	+ + +
						3rd August 1912 ...	+ + +
						4th August 1912 ...	+ + +

The "carrier" was a patient in the Cholera Hospital from 6th to 13th July, when he was discharged, he was arrested at Sakhigopal (near Puri) for bad livelihood and sent to Puri Jail on the 23rd July. He was released from Puri Jail and sent to the Cholera Hospital till 4th August, when he left Puri and I have no further record of him after that date.

This was a very interesting case. He was discharged "cured" from the Cholera Hospital, but like other convalescents he was still infective and was proved to be so nearly a month after his attack. He left Puri to return to his home and the bacteriological examinations ceased, but it is quite likely that he will remain infective for some considerable time as he no doubt harbours the Cholera vibrio in his gall-bladder.

Bacteriological examination of acute Cholera cases.—In the examination of the stools of the acute Cholera cases a culture of the Cholera vibrio was obtained on agar without preliminary enrichment in peptone water.

The following table gives the date of attack, the date of examination, and of recovery or death, the presence or absence of the Cholera vibrio in the stools of each of the acute cases of Cholera which occurred in Puri Jail during July and August 1912.

No.	Name.	Age.	Sex.	Caste.	Date of attack.	Date of recovery or death.	Date of examination of stools.	Presence or absence of cholera vibrio in stools.
1	C. H. ...	35	M	H	27th July 1912	14th August 1912	27th July 1912	+
2	B. M. ...	27	M	H	31st July 1912	10th August 1912	31st July 1912	+
3	D. U. ...	22	M	H	Ditto	Died 1st August	Not examined.	
4	B. D. ...	20	M	H	1st August 1912	9th August 1912	3rd August 1912	+
5	B. B. ...	23	M	H	2nd August 1912	14th August 1912	2nd August 1912	+
6	P. K. ...	26	M	H	4th August 1912	Died 4th August	4th August 1912	+
7	B. M. ...	19	M	H	5th August 1912	10th August 1912	5th August 1912	+
8	S. K. ...	20	M	H	6th August 1912	14th August 1912	6th August 1912	+
9	D. N. ...	18	M	H	7th August 1912	Ditto	8th August 1912	+
10	G. ...	22	F	H	Ditto	Died 8th August	Not examined.	
11	R. P. ...	12	F	H	8th August 1912	14th August 1912	9th August 1912	+
12	B. S. ...	45	M	H	Ditto	20th August 1912	8th August 1912	+
13	B. B. ...	28	M	H	Ditto	18th August 1912	9th August 1912	+
14	J. S. ...	52	M	H	9th August 1912	Died 9th August	Ditto	+
15	L. ...	58	M	H	10th August 1912	Died 13th August	12th August 1912	+
16	N. ...	30	M	H	11th August 1912	17th August 1912	Ditto	+
17	B. P. ...	16	M	H	12th August 1912	16th August 1912	13th August 1912	+

The strength at the time of commencement of the outbreak in the Jail was :—

Paid Officers and workers	29
Male convicts	189
Female convicts	4

It is significant to note that during the period of the outbreak there were several cases of "Diarrhœa" and "Dysentery." From the 24th July to 12th August 1912 :—

Number treated in Hospital	23
Number treated as out-patients	79

This epidemic is interesting from several points of view. In the first place because it was worked out on definite scientific lines which yielded valuable information for future guidance on several points. Further, it proved that the convalescent Cholera "carrier" is capable of causing an outbreak of Cholera. In the course of my investigation I demonstrated that a number of patients on leaving the Hospital harbour the Cholera vibrio and are still infective.[a] It might be asked is a convalescent "carrier" really capable of causing an epidemic, and is he of any great practical importance to the sanitarian in dealing with the Cholera problem? Such an experience as the present one, carefully worked out with strict methods of modern bacteriological research, affords a definite answer to these queries. It can hardly be regarded as an isolated or peculiar instance knowing as we do that many persons recovered from Cholera are still infective. This observation is important also as showing that this "carrier" was infected about a month after his attack; and on the last occasion I examined him he was excreting Cholera vibrios in his stool in very large numbers. After that he disappeared to his home and may continue a "carrier" for some time, and some obscure epidemics of Cholera which will trouble sanitarians to explain may follow his tracks. In view of my observations on the presence of the comma bacillus in the bile in Cholera,[b] it is probable that this man had an infection of his gall-bladder by the vibrio and large numbers of the organisms will be poured out into the intestine and so from time to time find their way into the external world in his stools.

Without such a bacteriological investigation it would have been impossible to have accurately traced the source of infection in this Jail epidemic; and its origin could only have been guessed at. Further, having appreciated the mode of origin and spread of the disease, it was possible to effectively control the epidemic by the application of methods of disinfection based on the researches.

The mode of production of this epidemic of Cholera is very similar to that of a number of outbreaks of Enteric fever which I had an opportunity of studying.[c] At that time there was a great paucity of scientifically ascertained facts regarding Enteric fever epidemics in India, so at the present time there is a similar absence of data obtained by modern methods of research in regard to Cholera epidemics. A belief which is very prevalent regarding the mode of dissemination of Cholera in India is that the disease is exclusively "water-borne." Undoubtedly water is a channel and an important one, but there are others, which, because they are less obvious, have not been taken sufficiently into consideration, but they are none the less important. It remains to be determined, by carefully made scientific observation, what the relative importance of the various means of dissemination of the Cholera virus in India is: there can be no doubt, however, as to the importance of the human factor in the spread of Cholera.

(*a*). Observation on Cholera convalescents and contacts. Major E. D. W. Greig, I.M.S.

(*b*). On the occurrence of the Cholera vibrio in the biliary passages and the pathological changes produced by it. Major E D. W. Greig, I.M S.

(*c*). Scientific Memoirs, Government of India. No. 32.

As I showed in my paper on Cholera convalescents and contacts in India, the daily bacteriological examination of the stools of acute cases of Cholera which occurred amongst the prisoners proved that at least two of them harboured the Cholera vibrio for long periods. Hence it will be seen that this "carrier" not only caused an epidemic of Cholera amongst the prisoners, but produced two Cholera "carriers" and these may become "permanent reservoirs" of the virus. In a free population where no precaution would be taken for the segregation of "carriers" produced by an epidemic, the situation is a very serious one. From the point of view of prevention probably the most dangerous issue of an epidemic of Cholera is the production of "reservoirs" of the virus. After each epidemic there must be a crop of "carriers," some "temporary," and a few more "permanent," which in their time, if suitable conditions offer will produce outbreaks of the disease in the same manner as the above described epidemic, and so the "vicious circle" of events continues. The problem, therefore, before the sanitarian is to find the weakest link in the chain and to endeavour to break it at that point. The problem is a very interesting one, but in the solution many practical difficulties will be met with, and will have to be overcome. I discuss this question in my paper 'On Cholera convalescents and contacts in Cholera.'

ALL-INDIA SANITARY CONFERENCE—MADRAS—NOVEMBER 1912.

OBSERVATIONS ON DISINFECTION IN CHOLERA

By

Major E. D. W. Greig, M. D., D. Sc., I.M.S. On special duty for the Cholera Enquiry.

I have recently had the opportunity in connection with my research work on Cholera of observing two instances of disinfection on a large scale carried out at my suggestion to arrest epidemics of the disease. As the plan of disinfection in both cases was based on the results of scientific research done during the progress of the epidemics of Cholera, and as the operations were watched and carefully controlled throughout the observations, I have thought it may not be without interest to place the results on record.

During the recent festival of Nutan Kalebar at Puri this year, a very large number of pilgrims assembled there in July 1912. It was estimated that not less than 300,000 persons were present at the Car pulling. Consequent on this large influx of people the annual epidemic of Cholera was much more severe than usual. In the first place there was the epidemic amongst the pilgrims, but later the town of Puri itself became infected and after the pilgrims left the local authorities of Puri had to cope with a second, and in view of the diminished population, a more severe epidemic during the month of August 1912.

Without entering into details of my researches on Cholera in Puri, which are dealt with by me in other reports, I may state that, broadly speaking, the factors which operated chiefly in the causation of the outbreak of Cholera were, (1) Convalescents discharged whilst still infective and closely associated with this, (2) Cholera "contacts", that is, healthy persons, who have been in contact with Cholera cases, and have acquired the infection without showing any signs of the disease, but they excrete the Cholera vibrio in their stools and are capable of infecting others. (3) Flies:—during the period of the festival enormous numbers of flies were present, amounting almost to a plague. I examined a number of flies taken near collections of Cholera cases and found that they were harbouring the Cholera vibrio on their external appendages, and also in their alimentary tract.

In regard to water I repeatedly examined samples from the sacred tanks, wells, protected and unprotected, etc., and although vibrios were present in some of the samples, none of these were Cholera vibrios, further the other features of the epidemic did not point to a water infection in the present outbreak.

At this point I would like to say that I have almost invariably found that there is a general impression that Cholera is entirely a "water-borne" disease; whilst in no way wishing to minimise the great importance of safeguarding water supplies in India, yet it is desirable, if progress is to be made, that a broader conception of the mode of dissemination of Cholera be taken. A very similar conception was held at one time regarding the mode of spread of Enteric fever in India, with the result that in spite of "vigorous sanitary measures" the disease increased instead of diminished.

I will proceed to discuss the two observations on disinfection.

(1) During the epidemic in the town, the Jail at Puri became infected by Cholera and as was shown by me the introduction of the infection was effected by means of a convalescent Cholera "carrier" who had been discharged from hospital a short time previously.[1] The investigation of this epidemic has been described by me in another paper.[2] In this communication I shall deal with the question of combating the epidemic by disinfection.

Seventeen cases of Cholera with 5 deaths occurred in connection with the Jail epidemic. The total strength of the Jail at the time of the outbreak was 222.

[1] An investigation of an epidemic of Cholera caused by a "carried". Major E. D. W. Greig, I.M.S.
[2] *Ibid.*

As the convalescent "carrier" had been in Jail a short time before he was isolated, the possibility, almost certainty, of the infection of the latrines, flies, and the production of "contacts" had to be remembered.

Having regard to my investigations during the present epidemic in Puri, I recommended the Superintendent of the Jail to commence at once a thorough disinfection of all fresh night soil in the Jail, in order that the specific organism might be destroyed in the stools of all within the Jail, as it was impossible to examine bacteriologically all the inmates to determine "contacts," and, secondly, to prevent the flies which were very numerous becoming infected. For this purpose Cyllin was used and each receptacle in the latrine was kept filled with a solution of sufficient strength and, if necessary, the stool was stirred up with the fluid to insure its being completely covered, which was an important point in order to guard against the possibility of fly infection. The systematic Cyllination was commenced on the 9th August and no fresh cases of Cholera occurred after the 13th August in the Jail.

The second table in the previous paper gives the date of entrance of the "carrier" into the Jail, and the daily number of admissions for Cholera.

Encouraged by this experiment the local authorities on my recommendation extended the disinfection to the whole town of Puri. At the time Cholera was present in a severe form. On the 13th August, 49 attacks and 39 deaths occurred. For the purpose of town disinfection I selected Chlorinated Lime because (1) it was cheap, (2) it is a very good bactericide, (3) and, very important, the pungent odour is most effective in keeping away the flies. Systematic chlorination of public and private latrines in Puri was commenced on the 16th August.

The following table shows the daily deaths from Cholera in Puri during the month of August 1912.

Date.	Death.	Date.	Death.
1st	34	16th	24
2nd	26	17th	26
3rd	22	18th	24
4th	24	19th	19
5th	1	20th	18
6th	22	21st	15
7th	19	22nd	9
8th	26	23rd	8
9th	32	24th	4
10th	32	25th	5
11th	35	26th	5
12th	35	27th	5
13th	39	28th	4
14th	33	29th	6
15th	34	30th	3
		31st	1

The following table shows the quantity of Clorinated Lime used for the disinfection of fresh night soil in the town of Puri.

Date.	Quantity.
August 15th	1 Cwt.
„ 16th	3¼ „
„ 17th	16¼ „
„ 18th	9¾ „
„ 21st	... ...
„ 22nd	3¾ Cwt.
„ 23rd	6½ „
„ 24th	9¾ „
„ 25th	9¾ „
„ 26th	9¾ „
„ 27th	13 „
„ 28th	6½ „
„ 29th	9¾ „
„ 30th	3¼ „
„ 31st	3½ „
September 1st	6½ „
„ 2nd	6½ „
„ 3rd	9¾ „
„ 4th	6½ „

The difficulties in carrying out the systematic disinfection were very great owing to the extremely defective state of the private latrines and the absence of proper access to them. It is to be hoped that this state of affairs will be remedied in the near future in order to facilitate the prevention of epidemics of Cholera at Puri. But in spite of these difficulties, the disinfection of fresh night soil of the town was followed by a very gratifying and marked fall in the number of daily attacks and deaths from Cholera, and in a fortnight the town became quite free from the disease. During the course of the disinfecting operations I visited the public and private latrines in Puri and was struck by the complete absence of flies round the receptacles although they were still very numerous elsewhere in the town; and this was extremely important having regard to the fact, that flies, as my researches showed, were active agents in the dissemination of the Cholera organisms in the present outbreak.

Comparison of the geographical distribution of cases, which occurred before and after systematic chlorination was carried out, shows that a very striking diminution in the number of fatal cases of Cholera in all the Shahis or Divisions of Puri town has taken place during the period of systematic chlorination of night soil. Even the Shahis—Harchandi Shahi and Basali Shahi—with the most defective latrine arrangements and in which many fatal cases of Cholera occurred responded to the treatment.

In combating both these epidemics the active factor, and indeed the only one which was brought into operation, was systematic disinfection of fresh night soil; in both instances it was followed by striking results.

The inhabitants of Puri appreciated the value of the operation and were so much impressed by it that the private house owners proposed to purchase Clorinated Lime and make use of it in their latrines during future periods of Cholera prevalence. At the time the disinfection operations were commenced there was considerable alarm in the town on account of the threatening aspect of the epidemic of Cholera: when it was seen that the operations were successful, the inhabitants who had gone away from Puri on account of Cholera began to return and complete confidence was established in this way.

The effect of pipe water supplies on the reduction of cholera in urban areas.

On inspection of Table I, showing the mortality from cholera in the towns of the United Provinces before and after the provision of a water-supply, the point that will at once attract attention is that cholera has persistently appeared nearly every year after the introduction of a water-supply except in the hill station of Naini Tal. The double vertical lines (||) in Table I delineate the period in which the water-supply schemes were completed and for three years after this date we should eliminate the cholera figures from any calculations we may make, as it always takes some time before the people appreciate the advantages of a pipe water-supply. These years have consequently been printed in Table I in antique figures.

On looking carefully into Table II the first noteworthy point is column B. The number of years in which cholera occurred after the provision of water-supply as compared with the previous years, is seen to be reduced only in the case of Cawnpore and Naini Tal. In Mussooree, Meerut and Agra it is actually increased.

Secondly, in column C, though the maximum mortality of any one year has been considerably lowered by the provision of water-supply, nevertheless a fairly high death-rate per mille has still occurred. Dehra Dun shows 2·25 after as against 10·19 before; Meerut shows 2·21 after as against 3·37 before; Benares 3·02 after as against 7·49 before; Lucknow 4·50 after against 4·39 before and Naini Tal 2·86 after against 10·19 before. On examination of column D, the number of years in which the cholera death-rate rose above 1 per mille is seen to have been reduced by the filtered water-supply to about half in Dehra Dun, Meerut, Benares, Lucknow and Naini Tal, and in Mussooree, Agra and Cawnpore to nil. Column E shows that notwithstanding occasional outbreaks and the increased number of years in which cholera has occurred in Mussooree, the effect of the provision of a protected water-supply has been to greatly reduce the average cholera mortality.

In attempting to explain the reasons why cholera has not entirely disappeared and the difference in the relative cholera mortality in various towns after the advent of a water-supply we have to enquire if—

(1) There are other sources of water, such as

(*a*) river and (*b*) wells, in addition to the pipe supply; and if these sources are largely used?

(2) What is the class of people inhabiting the town?

(3) Is the town a pilgrim centre?

(4) Is the pipe supply continuous or intermittent?

(5) Has the maximum mortality any relation to the prevalence of cholera in the district and consequently is it due to importation?

(6) Are there any other special circumstances bearing on the point?

(7) Have we sufficient data from which to draw conclusion?

I.—The towns situated on rivers are—

Agra, Cawnpore, Allahabad, Lucknow and Benares.

Agra has never had a mortality above 1 per cent. after the advent of water-supply.

Cawnpore in 1911 shows the rise to 1·84 and
Lucknow in 1901 ditto 4·50

Meerut is not near a canal or river. Naini Tal and Mussooree are hill stations, and near Dehra Dun is the Rajpur canal.

In Agra the river is near part of the thickly populated portion of the town. In Cawnpore the Ganges is some distance from the densely inhabited area, but a canal passes through the town. In Allahabad the Jumna is near the town, while the sacred river, the Ganges, is some distance away from the main portion of the city.

In Benares the whole city and temples practically front the Ganges and as is well known the river at this spot is considered particularly sacred. In Lucknow the Gumti is used for bathing purposes and is largely contaminated by sullage. In all these towns, with the exception of Lucknow, the river forms the part of the water-supply and is drunk by the inhabitants.

The question of wells as a source of water supply will be deferred until other points have been dealt with.

II.—What is the class of the people inhabiting the town ?

(1) Benares is inhabited by the most bigotted Hindus, faqirs, priests, &c., and is visited by pilgrims from all parts of India.

Agra, Cawnpore, Allahabad and Lucknow are largely populated by Hindus and Meerut practically by equal numbers of Hindus and Muhammadans.

	Muhammadans.	Hindus.	Christians.
Dehra Dun ..	8,983	19,018	1,460
Mussooree ..	1,443	3,494	1,550
Meerut	36,661	37,242	811
Agra	54,194	103,094	2,622
Cawnpore ..	41,834	111,359	2,790
Allahabad ..	47,934	105,834	4,932
Benares	55,407	142,756	981
Naini Tal ..	1,142	7,109	1,292
Lucknow	99,117	134,381	4,496

The effect of the larger Hindu population increasing the cholera death-rate is not apparent.

III.—Are any of these cities pilgrim resorts ?

Benares is a most renowned pilgrim centre and is constantly infected by cholera through importation. At Allahabad the important fair, the Magh Mela, is held once a year for a duration of about six weeks. This mela is under the very careful supervision of the Sanitary department.

IV.—The question of continuous or intermittent water-supply.

At Lucknow the supply is intermittent, being shut off from 8 p.m. *till 5·30 a.m.* In Meerut the water supply is continuous.

In Allahabad it used to be continuous but is at present intermittent. In Benares, Cawnpore, Agra, Mussooree and Naini Tal it is intermittent.

Water supplies are at present intermittent for several reasons.

Firstly, the waste of water in continuous water-supplies is enormous, the level of subsoil water is often raised and the general health deleteriously affected by malaria from surface pools where this method is in vogue. The introduction of the divisional meter system and the provision of cocks or stand-posts, allowing only a modicum of water to be drawn at one time, would obviate these drawbacks.

V.—Has the occurrence of cholera in a district any connection with a corresponding cholera high death-rate in towns, during the same season ?

It appears that in many cases when cholera is high in the districts it is also high in the towns as per example, the following :—

		Town.	District.
Allahabad ..	1906 ..	1·35	2·47
	1908 ..	1·41	1·79
	1910 ..	1·07	3·42
Benares ..	1903 ..	1·05	1·57
	1905 ..	1·03	1·65
	1906 ..	1·70	3·52
	1910 ..	1·50	5·14
	1911 ..	1·06	1·42

There is however no definite relationship between the amount of cholera in the district and that of the town. On the other hand there are cases in which the cholera rate was high in towns and low in the districts as seen in the following table :—

		Town.	District.
Cawnpore ..	1911 ..	1·84	·21
Meerut ..	1900 ..	2·21	·50
Benares ..	1896 ..	3·02	1·31
	1897 ..	1·69	·78
	1907 ..	1·18	·33
Lucknow ..	1909 ..	1·26	·75

and also cases in which it was high in the districts and low in the towns—

	Districts.	Town.
Agra, 1906	1·02	·35
Cawnpore, 1903	1·77	·56
„ 1908	1·14	·89
Allahabad, 1897	2·28	·43
„ 1902	1·22	·44
„ 1903	1·32	·38
Benares, 1899	1·13	·15
„ 1900	3·63	·77
„ 1910	5·14	1·50
Lucknow, 1906	1·10	·27
„ 1908	1·76	·85
Naini Tal, 1906	5·01	·66
„ 1909	1·17	·09

VI.—Are there any other circumstances bearing on this point ?

The hill stations of Mussooree and Naini Tal when compared show very different rates of cholera mortality. Before the water-supply the average death-rates were ·35 and 2·26 and the maximum mortality 2·92 and 10·19 respectively. Whereas after the water-supply the average cholera death-rate was ·22 and ·44 and the maximum ·51 and 2·86 respectively. The water-supply of Naini Tal has been shown in a paper read by Captain Dunn on the working of the Jewell Filter to be contaminated by bacilli coli especially from the Pilgrim springs.

The springs at Mussooree are from a deeper source or are less exposed to surface contamination before exit. The food supplies of Naini Tal come from the Tarai and Bhabar, a notoriously bad place for cholera. The coolies for Naini Tal come from Kumaun and Garhwal and the Mussooree coolies are mostly from Tehri Garhwal and the higher elevation of Garhwal (the lower parts of Garhwal occasionally suffer from severe outbreaks of cholera).

One explanation for the more frequent attacks of cholera in Mussooree is that the famous pilgrim resort of Hardwar is on the Dehra Dun railway line which was opened in 1900 and from Hardwar many pilgrims who formerly proceeded by Lachman Jhula now pass through Mussooree *en route* to Gangotri and Jamnotri, the sacred sources of the Ganges and Jumna, and often bring cholera in their train. This sufficiently explains the increased frequency of cholera at Mussooree after 1900.

At Dehra Dun at an elevation of about 2,300 feet above the sea level the water-supply was originally from the hill streams and the Rajpur canal which were liable to contamination along their whole course. The maximum cholera mortality in one year was as much as 10·19. After the opening of the water-supply the average cholera death-rate dropped from 1·91 to ·55 but the source of Dehra Dun water-supply is not as yet sufficiently protected. At one point the water from springs is allowed to flow in uncovered channels to a collecting basin instead of being built over. The occasional outbreaks at Dehra Dun are also probably commenced by pilgrims from Hardwar, proceeding to the holy places in the hills beyond Mussooree.

Though not affecting the incidence of cholera to any appreciable extent, the following story, the truth of which is vouched for by Mr. Lane Brown, who was Municipal Engineer of Benares for six years, will show one of the many ways in which our Aryan brother is exposed to cholera and other diseases through his water-supply.

A water carrier was noted to take up a handful of street mud and place it in his water vessel which he had just filled from the street tap. He was asked why he did this and the burden of his reply was that certain orthodox Hindus required Ganges water to drink but he was not going to bother to go to the river so he gave them filtered tap Ganges water and a little street mud to replace that which had been removed and which his clients considered indispensable to its sanctity.

The relation of the wells in a city to cholera should now be investigated.

On reference to Table III it will be seen that the number of public wells and private wells in the towns of Cawnpore, Allahabad, Lucknow and Benares is large while the number in Agra is comparatively small. It will be noted from Table I that the cholera rate in Agra has never been above 1 per mille, whereas in Cawnpore

it has occurred once, in Allahabad thrice, in Lucknow four times and in Benares eight times. On eliminating the 1906 outbreak of Allahabad at the Kumb Mela and the fact that cholera is mainly introduced during the mela periods we find that the number of years in which the cholera mortality has risen above 1 per mille bears a close relation to the number of wells in the city.

Under the Municipal Act, though powers have been given for the protection of public wells, private wells cannot be dealt with and may be situated within 1 foot of a latrine.

In Mandalay the author has seen a well used as a latrine with a seat constructed over it, and although the drinking water-supply was taken from a well several yards away the sub-soil water was directly polluted.

Under the building bye-laws when adopted, we shall be able to prevent the erection of a latrine near an existing well, but no law can stop a house owner from digging a well near his latrine. Indeed so common has this become that in Farrukhabad-cum-Fatehgarh the high death-rate 80·71 has been ascribed by a facetious District Magistrate to the well-cum-latrines which abound in the houses of the town. In a town with a water-supply one naturally wonders why wells should be used at all. The objection of the Hindus to taps and a pipe water-supply is being gradually overcome, but in some cases in the event of a cholera outbreak he immediately puts the disease down to the pipe supply. Sterility and incompetency are also put down to drinking from pipe water-supply which, as the Indian points out, is not as good for his garden as is an unfiltered supply, and if not good for garden produce is naturally not good for himself, and lastly, but not leastly, the pipes of the filter water supplies are so exposed to the sun that drinking practically hot water from the tap does not appeal to any one's palate, when comparatively cool water from a well is near at hand. As a result of the heat in pipe water the practice has been adopted of putting tap water into gharas and placing them in the dark to cool. No covers are placed over these gharas and there is a tendency to their infection by dust and to the production of mosquito larvae.

Mr. Baldwin Latham in providing a pipe water-supply to a city always placed the mains 5 feet below the crown of the road, as (he stated) the temperature was kept lower and the infantile mortality in hot weather was reduced.

In India the water is subjected to great heat in the filter beds and in the clear water reservoirs. When raised into iron balancing tanks it becomes extremely hot. The mains are only 3 feet below the ground surface and the supply pipes running up the sides of the house are unprotected from heat, with the result that the water is delivered often so warm as not to require heating for the evening bath.

In Table IV in which the heat in stand pipes, shallow and deep wells is compared, there is not a very marked difference at this time of the year, and the temperature recorded are not reliable. In Table VI the temperature of pipes and springs at Mussooree and Naini Tal are compared. It is unfortunate that investigations on the subject were only commenced in September and we have no record of the heat in May and June. Table V shows the heat in the author's room in Lucknow situated in the third storey in which the tap temperature in September reached 98.

In Lahore, with several balancing tanks, the supply in the hot hours of the day is less than at any other period of the 24 hours. In Lucknow the Water Works Engineer, whose office is under the raised reservoir, states that the water is undrinkable in the hot weather and is too hot even to bathe in.

The protection of the water-supply from heat is the problem I am bringing forward for discussion to-day, in the hope that some method may be found to reduce this temperature of the water in our taps, not to iced coldness, but to the temperature of the water of a shallow well. To place the main pipes 5 feet under ground and protect the pipe connection from direct sun and heat might be an improvement, but how are balancing tanks to be dealt with unless they can be converted into thermos flasks, or covered with asbestos, eternite sheets, sawdust, mica or some non-conducting material? It should be possible for filtered water reservoirs to be kept cooler. Some are only covered by a few inches of soil.

The powers given by the Municipal Act over wells deals in every case except in section 126, with public wells and water supplies.

Section 110 simply gives power to close or replace any wells if they are dangerous from want of repair to persons passing by or dwelling or working in the vicinity.

Section 119 deals with a suitable place set apart for bathing and washing clothes and gives no jurisdiction over private wells.

Section 126 requires the owner of any well to prevent access to it and to prohibit the removal or use of the water for drinking if the Civil Surgeon or the Health Officer considers that the water may engender or cause the spread of any dangerous disease. This section can only be enforced in a municipality after it has been applied to the municipality by the Local Government.

Section 128 (IV) gives the board power to make rules for supervision over and regulating the use of public streams, springs, tanks and wells and other sources from which water is or may be made available for public use.

Section 93 prohibits filth receptacles being placed within 50 feet of spring, well, tank, reservoir or other source from which water is or may be supplied for public use.

It is impossible to compel a house owner with a site of perhaps 40 feet long to put his latrine 50 feet from his own or his neighbour's well but to permit a well, to be within a few feet of a latrine and to allow urine and kitchen water drains to pass outside the cylinder, appears to require some action which at present can only be taken in the case of lodging houses by prohibiting the issue of the licence until the condition of affairs is altered.

Further, the site plans of houses are constantly being submitted in which the well is 2 feet from the latrine. These plans are not passed unless the latrine is a connected privy. What action can be taken if after the erection of the house the owner sinks a well next to its latrine? The answer is *none*! In other words we protect the public but permit the private individual to do the most insanitary things and have the most insanitary arrangements in his house, with the result that the private individual of which the public is composed thinks your measures a farce and they are only introduced as a means of *zabardasti* or oppression.

One solution of the difficulty is apparently perfectly simple. We are dealing with conditions in sewered towns. Why not compel the owners of houses within 100 feet of a sewer to link up and to put in connected latrines, &c.?

Perfectly plain sailing in theory but let us examine the working in practice. Section 20 of the Sewerage and Drainage Act reads as follows:—

"When any building or land situated within one hundred feet of a municipal drain is at any time not drained to the satisfaction of the municipal authority by any, or a sufficient drainage connection with such drain, the municipal authority may by notice require the owner or occupier of such building or land to make and maintain a drainage connection with the drain in such manner as the municipal authority may subject to rules made under section 33, sub-section 3, direct."

In Cawnpore a sewer costing 10 or 12 lakhs has been completed for the last six years. The board having been given permission under the word "may" to do as it likes has at last passed a bye-law to the effect that any house newly erected, costing over Rs. 2,000 and situated within 30 feet of a drain sewer, shall connect up. After six years and the expenditure of 10 lakhs 30 connections of which 5 only were constructed last year have been made and a bye-law passed as above. A truly magnificent effort, and one that should conduce the thoughtful members of the community to wonder whether the word "may" in the Act should not be changed for "shall" or whether boards should not delegate certain of their powers to the Chairman or Health Officer, to relieve them from the constant trouble of importunate and apparently conscientious objectors against the working of an Act which would compel the conscientious objectors to spend a little money on much needed improvements.

Here again we are met with difficulties; the cost of putting in a flushing latrine and connections is not small from a poor man's point of view. An intermittent syphon flush causes a waste of water. Can any of my hearers recommend a cheap, useful, self-flushed native seat for the poorer inhabitants of private houses, cheaper than the Donaldson's seat ?

Finally, deductions from such insufficient data as have been produced in this paper must be considered in the highest degree unsatisfactory, and yet the fact remains that cholera has not been eliminated from these towns, and if we waited before suggesting methods of improvement based on general knowledge until definite proof was obtained, the author would be dead and thousands of human beings who might have been saved would have entered the bourne from which there is no return.

The lessons to be learnt are—

(1) that the unfiltered water supplies of towns must be sufficiently protected at their source, and

(2) that wells will continue to be a source of danger until the pipe water-supply can be protected from excessive heat. With regard to control over private wells, we do not seek to eliminate them, as the country is not ready for such a step nor is our pipe supply sufficiently protected from heat to render this course possible.

But we do ask for the means of enforcing the protection of private wells in sewered towns by linking up latrines and house drainage with sewers. In unsewered towns permission to construct a well in a house should be obtained from the Chairman, Municipal Board. The Health Officer should report on the local conditions and get a tube well put down when the sub-soil water level will allow it. Where this is impossible the well should be placed as far as is possible from the latrine and its drains, which should also be constructed of approved pattern and put into thorough repair before permission to sink a well is granted.

There are thus three points for consideration and discussion :—

(1) The reduction of temperature in pipe water supplies.

(2) The provision of further powers to protect private wells.

(3) The type and cost of a suitable connected native pattern privy seat and the approximate cost of connection with a sewer 50 feet distant.

TABLE I.

Comparative statement showing death-rate per mille of population from cholera in the larger municipalities with water-works and the districts excluding such municipalities, United Provinces, for 31 years.

Number.	Larger municipality with water works and district excluding municipality.	Date of introduction of water-works.	1881.	1882	1883.	1884.	1885.	1886.	1887.	1888.	1889.	1890.	1891	1892.	1893.	1894.	1895.	1896.	1897.	1898.	1899.	1900.	1901.	1902.	1903.	1904.	1905.	1906.	1907.	1908.	1909.	1910.	1911.
1	Dehra municipality	1896, March 12th	..	..	..	..	·89	..	2·68	..	..	7·07	·05	10·19	..	·04	·04	3·24	..	..	**2·70**	..	..	·17	2·25	..	..	·83	·46	·12	·82	1·69	·23
2	Mussooree ditto ..	1895, April 15th	..	..	·32	..	·32	..	..	..	..	·97	..	2·92	..	..	..	**·19**	**·13**	..	·13	·13	·41	·51	·31	..	..	·51	..	·20	·20	·41	..
3	Dehra Dun district	..	·02	..	..	·24	·14	..	1·45	·05	..	1·82	·08	19·92	·07	·01	..	1·01	..	..	·98	..	..	·15	5·20	·08	..	·98	·08	·11	·70	·58	·24
4	Meerut municipality	1896, May 6th..	·41	·02	·01	·19	·04	·01	2·29	..	·08	..	3·37	·30	..	..	·04	·05	**·01**	**·01**	**·13**	2·21	·06	·08	·76	..	·01	·93	..	·16	·61	·01	·09
5	Meerut district ..	..	·20	·01	·01	·09	·02	..	1·29	..	·42	·10	·51	2·68	·05	·01	·02	·25	..	..	·16	·50	·05	·27	·37	·02	·03	·05	..	·12	·06	·02	·11
6	Agra municipality..	1890, December 3rd.	·06	·02	2·58	3·74	·21	..	2·03	·03	·74	2·49	**·04**	**·33**	**·01**	·13	·01	·22	·67	·03	·03	·06	·67	·04	·49	·01	·01	·35	..	·02	·04	·01	·03
7	Agra district ..	..	·01	·08	·57	·67	·60	·01	·87	·01	2·35	·95	·02	1·24	·01	·10	..	·08	·98	·01	..	·41	·25	·37	·38	..	..	1·02	·08	·52	·07	..	·01
8	Cawnpore municipality.	1894, March 17th.	·04	·77	·85	1·25	·47	1·61	2·77	·02	·57	·04	5·35	1·05	·06	5·83	**·68**	**·40**	**·57**	·02	..	·07	·74	·05	·81	·05	..	·56	·07	·89	·93	·45	1·84
9	Cawnpore district ..	..	·06	1·35	·52	·39	·86	·25	4·93	·05	·09	·01	·68	·59	..	4·67	·22	·02	·65	·01	..	·08	·62	·04	·20	·02	·01	1·77	·25	1·14	·12	·05	·21
10	Allahabad municipality.	1891, March 26th.	·24	1·21	·91	·14	1·43	·01	2·80	·62	·60	·43	1·12	**1·39**	**·07**	**2·80**	·11	·60	·43	·03	·01	·57	·24	·44	·38	·04	·18	1·35	·63	1·41	·07	1·07	·67
11	Allahabad district..	..	·18	1·02	·37	1·01	2·24	·10	5·15	·13	1·30	·23	5·77	3·88	·02	1·87	·34	·73	2·28	·01	·02	·79	·25	1·22	1·32	·06	·91	2·47	·11	1·79	·11	3·42	1·02
12	Benares municipality.	1892, November 18th.	1·71	3·64	2·38	2·43	·83	1·15	7·49	2·74	1·22	·58	2·67	2·29	**·72**	**1·36**	**1·10**	3·02	1·69	·15	·15	·77	·87	·87	1·05	·74	1·03	1·70	1·18	·88	·14	1·50	1·06
13	Benares district ..	..	·68	2·03	1·11	·27	·83	3·20	4·40	1·07	1·21	·24	3·16	1·36	·34	3·24	1·52	1·31	·78	·03	1·13	3·63	·45	·72	1·57	·07	1·65	3·52	·33	·16	·69	5·14	1·42
14	Lucknow municipality.	1894, July 21st	·59	2·76	·72	·76	3·82	1·17	4·39	·07	·40	2·97	3·55	1·67	·08	3·43	**·09**	**1·49**	**1·90**	·08	·03	·86	4·50	·06	·10	·10	1·72	·27	·12	·85	1·26	·32	3·41
15	Lucknow district...	..	·39	2·48	·38	·26	1·05	·79	9·31	·11	·88	1·05	6·66	3·77	·01	5·96	·40	·17	2·09	..	..	·37	2·61	·44	·48	·03	3·31	1·10	·30	1·76	·75	·66	8·83
16	Naini Tal municipality.	1892, October..	·15	..	..	·30	..	·30	2·28	..	7·45	10·19	·68	5·83	..	..	..	·76	2·86	..	..	..	..	..	1·02	..	..	·66	..	..	·09	..	..
17	Naini Tal district ...	..	·14	·23	..	·92	·10	·05	4·69	..	14·20	1·11	1·63	7·06	·0.	·26	..	4·17	·50	·03	·09	..	·23	1·84	1·97	·02	·01	5·01	·67	1·13	1·17	·81	·60

TABLE II.

Serial No.	Name of place.	A. Number of years examined.		B. Number of years in which cholera occurred.		C. Maximum mortality.		D. Number of years in which the death rate per mille was above 1.		E. Mean death rate per mille (annual).		Remarks.
1	Dehra Dun Municipality	12	*12*	8	*8*	10·19	*2·25*	4	*2*	1·91	*·55*	Figures entered in Roman appertain to the period before the introduction of water works.
2	Mussooree „ ..	13	*13*	4	*9*	2·92	*·51*	1	..	·35	*·22*	
3	Meerut „ ..	12	*12*	8	*10*	3·37	*2·21*	2	*1*	·53	*·41*	
4	Agra .. „ ..	10	*10*	9	*10*	3·74	*·67*	4	..	1·19	*·23*	
5	Cawnpore „ ..	13	*13*	13	*11*	5·35	*·93*	4	..	1·14	*·36*	Figures entered in *italics* appertain to the period after the introduction of water works.
6	Allahabad „ ..	10	*10*	10	*10*	2·80	*·60*	3	*3*	·84	*·28*	
7	Benares . „ ..	12	*12*	12	*12*	7·49	*3·02*	10	*6*	2·43	*1·10*	
8	Lucknow „ ..	14	*14*	14	*14*	4·89	*4·50*	8	*4*	1·88	*·98*	
9	Naini Tal „ ..	12	*12*	8	*4*	10·19	*2·86*	4	*2*	2·26	*·44*	

TABLE III.

Number of wells in large towns with a piped water-supply.

No.	Place.	Population.	Rivers.	Total.	Wells.					
					Number of private wells.			Number of public wells.		
					Drinking.	Washing.	Total.	Drinking.	Washing.	Total.
1	Cawnpore ..	173,973	Ganges	2,559	2,097	119	2,216	320	23	343
2	Lucknow ..	240,016	Gumti	4,600	..	..	3,715	..	..	885
3	Allahabad ..	159,701	Ganges and Jumna	1,258	..	..	606	..	..	652
4	Benares ..	199,868	Ganges	3,252	518	1,899	2,417	830	5	835
5	Meerut ..	76,351	..	Not known	235	..	..	6	..	..
6	Agra ..	163,935	Jumna	445	170	235	405	13	27	40
7	Dehra Dun ..	30,301	..	..	..	..	..	..	..	..
8	Mussooree ..	*11,986	..	..	..	..	..	..	..	..
9	Naini Tal ..	*13,183	..	..	..	..	..	..	..	..

* Mean between summer and winter population.

TABLE IV.

Temperature in wells and stand-pipes in September and October 1912.

Serial No.	Place.	Deep wells.				Stand pipes.				Shallow wells.				1912.	
		7 a.m.	12 a.m.	3 p.m.	6 p.m.	7 a.m.	12 a.m.	3 p.m.	6 p.m.	7 a.m.	12 a.m.	3 p.m.	6 p.m.	Month.	Date.
1	Allahabad (Centigrade)	26·4	27	27	27	29·4	29·8	29·4	29·2	27·6	27·8	27·8	27·6		
	Allahabad (Fahrenheit)	79·5	80·6	80·6	80·6	84·9	85·6	84·9	84·3	81·7	81·7	81·7	81·7		
2	Cawnpore .. Ditto ..	79	79	79	77	88	88	88	81	76	79	79	77		
3	Benares (Centigrade)	25·3	26	26·2	24·3	26·2	28·3	29	27·2	24·4	26·2	25·1	25·2		
	Benares (Fahrenheit)	77·5	78·8	79·1	79·3	79·1	82·7	84·2	80·7	76	79·1	77·1	77·3		
4	Lucknow	72	72	72	72	78	73	74	76	76	72	74	74	October ..	25th
	Do.	70·8	80	78·0	80·1	80·2	80·4	80·4	80·4	70·8	80·4	80·0	80	September	25th

(9)

TABLE V.

Temperature of water in the Sanitary Commissioner's quarters (Lucknow), third storey.

Date.	7 a.m.	12 a.m.	3 p.m.	6 p.m.
	F.	F.	F.	F.
September 1912, 22	84°	91°	93·5°	90°
„ „ 23	84°	96°	97°	90°
„ „ 24	86°	98°	98°	94°
„ „ 25	86°	92°	92°	90°
„ „ 26	85°	89°	90°	84·5°
„ „ 27	81°	90°	89°	88°
„ „ 28	83°	92°	90°	86°
„ „ 29	87°	89°	91°	88°
„ „ 30	88°	94°	93°	90°
October „ 1	89°	97°	96°	92°
„ „ 2	92°	98·5°	93°	92°
„ „ 3	85°	97°	98°	92°
„ „ 4	86°	96°	94·5°	91·5°
„ „ 5	82·5°	90°	94°	86°
„ „ 6	84°	93°	90°	85°

TABLE VI.

Water temperatures at Mussoorie.

No.	Name of place.	7 p.m.	12 Noon.	3 p.m.	6 p.m.
		F.	F.	F.	F.
1	Murray spring No. 3 ..	62°	61°	62°	61°
2	Reservoir at Pump House ..	62°	63°	63°	63°
	Stand pipes.				
3	Herne Dale	54°	64°	65°	64°
4	Maple Hayes	52°	64°	64°	64°
5	Below kutcheri	64°	64°	64°	64°
6	End of Landour Bazar ..	64°	64°	64°	64°

Water temperatures at Naini Tal.

No.	Name of place.	8 a.m.	12 p.m.	3 p.m.	5 p.m.	6 p.m.
		F.	F.	F.	F.	F.
1	Tank near Ardwel ..	60°	62°	63°	63°	63°
2	Standpost in New Market ..	62°	66°	66°	64°	64°
3	Parsonage Tank ..	62°	66°	66°	65°	65°
4	Armadale Tank	60°	62°	63°	63°	63°
5	Forest Spring No. ii ..	58°	60°	60°	56°	54°
6	Cheena Spring	56°	58°	56°	55°	55°

ALL-INDIA SANITARY CONFERENCE—MADRAS, NOVEMBER 1912.

SOME PRACTICAL POINTS IN DEALING WITH EPIDEMIC OF CHOLERA.

BY

MAJOR H. A. F. KNAPTON, I.M.S., ACTING SANITARY COMMISSIONER FOR THE GOVERNMENT OF BOMBAY.

Of the three common infectious diseases with which we are chiefly concerned in this country, occurring as they do, from time to time in epidemic form, *viz.*, Small-pox, Plague and Cholera, I think it will be unanimously admitted that we are better acquainted with the nature and means of dissemination of the last than of the other two, hence we are better able to devise methods for preventing its spread.

We know little or nothing of the active cause of small-pox, we can only infer from comparison with other complaints of the kind that it is due to the introduction into the body of some virulent organism, which after running a definite course of incubation followed by certain pronounced physical lesions is cast off mostly by the skin, if the patient survives long enough.

From experience we know that the malady may be contracted by merely coming into contact with an infected individual, or even residing near him, it may also be conveyed through the medium of cloths, bedding, etc. Wherefore our only means of preventing its spread is by isolation of the patient, and vaccinating all those who have been in any way associated with him, with a view to rendering them immune, at the same time disinfecting everything which has been used in the sick room.

We know that plague is due to the invasion of a specific microorganism which we can cultivate outside the body, and there is every evidence to show that it is imparted to human beings through the medium of a certain type of parasites found on rodents, but we are still more or less in the dark as to the exact means by which it is brought about. Wherefore we organise campaigns for the destruction of rats, inoculate with immunising serum, and encourage people to evacuate the houses in which it appears.

But when it comes to cholera we are practically certain that the disease is caused by toxins produced in the body during the growth of a certain vibrio, having definite characteristics, whose habitat is in the alimentary canal and that it is invariably introduced into the system by the mouth, almost always either directly or indirectly in infected water it may be actually present in drinking water which is no doubt the commonest way people contract the disease. Or it may be that vessels or utensils used for feeding purposes have been washed in contaminated water. Soda water bottles may have been kept in a *chatti* containing infected water, a little of which is retained in the necks above the glass ball stoppers and mixes with the contents when they are opened, the acidity being much reduced by the ebullition of the Co_2 and not having time to affect the microbes.

Green vegetables may become carriers of the malady either from being washed in water containing the organism after they are gathered, or from the ground in which they are cultivated being irrigated by water drawn from a suspicious source, moreover it is possible that the soil in which they are grown may have been directly polluted by a person who is suffering from cholera defecating upon it.

I think we can eliminate the theory of air borne infection, we know that the bacteria are not very tenacious of life, that they soon become inert and die when deprived of moisture or exposed to a bright sunlight, so we may be fairly certain that if they ever became so dried up to the extent that they cannot adhere to the

surface on which they are lying, and therefore liable to be blown about by the wind, they would have lost their vitality and be rendered harmless.

But there is one agency by which the disease may be disseminated, quite irrespective of water in any form which is very important and that is the part played by insects. The common house fly with his gummy plantar surfaces is undoubtedly a serious offender in this respect. Having settled on a cholera stool where a certain number of infective microorganisms stick to his feet, he goes off and gets drowned in a milk jug or samples some delicacy at a sweetmeat stall incidentally relieving himself of a few hundred bacteria here and there while crawling over it. And lastly we must not forget that infection may be conveyed by servants, who in this country are by no means over particular in the matter of cleanliness, in preparing or handling food. I believe not long ago an outbreak in a Calcutta hospital was traced to a ward-boy, the bacteria being actually found on the palms of his hands.

Our first object then must be to find out the source from which the disease originated, and then to prevent its further spread, either by rendering the source inert or by stopping persons putting themselves in the way of infection from it. Needless to remark, the latter presents considerable difficulties.

In the Central Division of the Bombay Presidency as soon as news reaches the Deputy Sanitary Commissioner's Office that a case of cholera has occurred in a village, a paper containing certain questions is sent there to be filled up by the *Kulkarni* (or village scribe). By this means we gain possession of certain particulars with regard to the patient's surroundings when he contracted the malady. The age, sex, position in life, what water he was in the habit of drinking, whether he was a permanent resident of the place or had lately come from some other neighbourhood, if any other member of the household had been similarly affected, etc., from which we can draw fairly accurate deductions, firstly, as to whether it was really a case of cholera or not, and secondly, whether the source of infection is actually present in the village or the disease has been introduced from outside.

An isolated case occurring in a house where the members are all living together under exactly similar conditions, none of them having lately been away from home, is hardly likely to be true cholera.

And here I may mention that we sometimes get a history of a man, who after eating a quantity of water melons while working in the fields all day, during the rains, has been taken with violent choleraic symptoms in the evening with perhaps fatal results. A case of the sort was published only a few months ago in the Indian Medical Gazette, the author asking if such phenomenon was within the experience of any other medical man.

During severe epidemics of course it is next to impossible to discriminate between the true disease and severe diarrhœa, possibly caused by ptomaine poisoning or some other violent irritant, from the accounts received; and the village officers knowing that they will be severely dealt with if they neglect to report an outbreak, take care to err on the safe side and enter any complaint which is accompanied by profuse evacuations and collapse in their cholera returns.

Last year in a Deccan village during a period of three or four days more than a hundred of the inhabitants developed symptoms of this kind, which lasted only a few hours and then disappeared entirely, no death occurred although the very old and infants in arms were equally affected, which at once aroused a suspicion that it could not be really cholera. And no cases of the sort were reported from other places in the vicinity, with which there was frequent intercourse.

It was all over before any systematic investigation could be made, and we never came to any satisfactory conclusion as to the real exciting cause, but it was evidently not cholera, although it was returned as such.

In addition to these queries a pamphlet in vernacular is forwarded entitled "Simple instructions to check the spread of cholera" and the *Mamlatdar* (chief revenue subordinate, I believe he is analogous to the Tehsildar of the Punjab) is requested to see that they are carried out.

I will not inflict a detailed transcript of these on you, suffice it to say that they explain that the disease is chiefly communicated by drinking water, hence great care should be taken to protect the general supply from pollution. They emphasize the necessity of observing personal cleanliness on the part of those attending the sick, and warn them of the virulently infective nature of the stools and in this connection, with your permission I will quote direct from the pamphlet.

"A fire should constantly be kept alight, and all stools and vomited matter should at once be burnt ; this is very important."

There is a paragraph warning persons of the danger of eating any food or drinking any milk with which flies have come in contact, and some advice regarding abstinence from unripe fruit, particularly water melons.

I believe this injunction leads to considerable misapprehension. Of course its main object is to lessen the probability of persons lowering their vitality by the diarrhœa which often follows indulgence in large quantities of indigestible matter, hence rendering them more likely to fall victims to the prevailing complaint. But quite a large proportion of the lay public imagine that cholera can be produced merely by eating fruit.

Now it is comparatively easy to frame a complete set of directions for use, so to speak, but the difficulty is to get them put into practice. And I may admit at once that the success or otherwise of these preventive measures almost entirely rests with the *Mamlatdars.*

If he is an intelligent man, with previous experience of epidemics and has confidence in them himself, he will often contrive to nip the disease in the bud. If on the other hand he is an ignorant individual, without any progressive tendencies, and regards our efforts merely as some new fad on the part of the ever incomprehensible *Sahib-log*, he does not trouble to give any orders on the subject. The village officers take their cue from him, the pamphlets become as valuable as any other waste paper, and cholera spreads unchecked from village to village throughout the country.

To show what can be done with the co-operation of an intelligent subordinate who has influence with the people, I would cite the case of Wai in the Satara district. This is a small town of a few thousand inhabitants, the head-quarters of Brahmanism in the Western Presidency, in so much that the Holy Krishna river runs through it. It is situated at the foot of the hill station, to which Government repairs in the hot weather; cholera appeared there at the end of May just as the general exodus to Poona was about to take place, some thirty cases occurring every day, and the cart-men employed to bring down baggage contracted the complaint on their way through the place and died on the road later. This was a very serious matter and called for immediate remedy. The *Mamlatdar* had a couple of thousand copies of the pamphlet already referred to printed at the local vernacular press and circulated amongst the inhabitants, at the same time exercising personal supervision to see that the advice contained therein was acted upon. He even went counter to the ancient prejudices of the people to the extent of having the sacred river patrolled by police to prevent the water being used for domestic purposes; it was undoubtedly contaminated with the specific microorganism.

The severity of the epidemic began to abate at once. The only thing he did not do was to see that the stools of persons suffering from the disease were burnt, but had them buried instead. Now this is not a safe-method of disposing of them, and for this reason I quoted the paragraph in the pamphlet referring to the subject, verbatim, just now. First of all their infectivity is not destroyed by merely putting them underground, and to save themselves trouble, people will deposit them in the softest and most porous soil, without any regard to the proximity of wells or other sources of drinking water, to which they may gain access later.

Furthermore the ground round many of the houses is so hard that it would take a pick to break it up, wherefore it is physically impossible to bury the excrement, and the usual alternative is to throw it away on the nearest convenient patch

of waste ground, whence it may eventually be washed into a neighbouring stream, and in the meantime is the general rendezvous for all the flies in the vicinity.

There is always sufficient *cutchera* lying about in every village with which to make a fire, and nothing is simpler than to have a few handfuls gathered, and when well alight pour the excreta on to it, at the same time holding the *gumlah*, or whatever vessel they were collected in, in the flames for a short time to sterilise it.

I visited Wai when the disease had been prevalent there about a week during which time there had been over a hundred attacks and some fiftyfive deaths and although the daily incidence had been diminishing owing no doubt to the preventive measures which had already been taken, as soon as this method of dealing with the motions was put into practice the epidemic stopped dead, only two cases occurring during the ensuing week.

Last year cholera broke out in Sholapur, a town having a population of nearly 100,000 persons, the centre of a great cotton industry. The water supply is brought into the place in an open channel from a large bunded tank three miles away. Knowing what we do of the habits of the natives of this country this means of conveyance always appears to me to be attended with considerable risk, particularly as no very active precautions are taken to prevent the populace gaining access to the water. There is what amounts to a public path, on either bank, with inhabited dwellings a few yards off, and it is crossed in places by plank bridges, one of which is the general means of communication with a village fifty yards away from it, wherefore it was of the first importance that the disease should not spread to this village or to any of the houses situated along the course of the conduit, as if the stream became infected nothing could avert the most appalling epidemic.

During the latter part of August twenty or thirty attacks were being reported daily. They were mostly confined to the lower portions of the town where the houses are very squalid and there is a certain amount of overcrowding, although a few cases occurred in other quarters.

There was a question of establishing a temporary hospital and insisting that all persons suffering from the complaint should be taken to it for treatment, without consulting their own inclinations in the matter.

Now this is a step which should never be resorted to, if it can possibly be avoided, as the majority of natives have a rooted antipathy to leaving their homes when they are ill; moreover they look upon it as a reflection on their domestic arrangements, hence such a procedure is a direct incentive to conceal the cases, which is the last thing desirable.

The people of the infected portion of the city drew their water supply from wells, which were carefully protected from pollution by the usual methods, a police guard being mounted at each to see that the precautions were not neglected.

A special sweeper was told off to each case, or group of cases, if two or three happened to be near one another in one street, they were provided with wood and made little fires in selected spots. Directly one of the sufferers passed a motion it was handed over to the sweeper on duty and burnt then and there. Phenyle was distributed to those attending the sick with instructions to wash the floor or any furniture soiled by the excreta, cotton clothes worn by patients were burnt, the municipality giving compensation for the loss of it in the case of the very poor, blankets and woollens were dipped in phenyle. The same old pamphlet was circulated in large numbers and pasted in conspicuous positions all over the city. The epidemic entirely disappeared in ten days. It is hardly necessary to tell you that it is far more difficult to carry on a campaign of this sort in a large city like Sholapur, than in a small town or village.

A few months ago cholera appeared in a village about three miles from Poona Cantonment, some thirty cases occurred before we knew anything about it, the official report having been returned by the *Mamlatdar* for correction. The same means were employed, the people being shown how to render the

excreta harmless by pouring them on a fire. They seem rather to enjoy the process as having some mystic signification. No further cases occurred.

To quote as instance of how the lower classes dispose of virulently infective dejecta when left to themselves. There was a woman in this village who when asked what she did with them, proudly answered that she threw them into the *nala*. The *nala* in question is an irrigation channel largely used for drinking and other domestic purposes, it flows round the village, her house being one of the furthest up stream. And with reference to this I would remark that it is with the women we have the greatest difficulty, the majority of them are so intensely stupid and obstinate, that even if they can be made to understand what is required of them, they will not do it. At Wai one old dame had a regular stand up fight with her son, a comparatively enlightened individual, when she was told not to drink the river water, to which she had been accustomed all her life, the police had to be called in to separate the parties.

In these three outbreaks, Gentlemen, I personally supervised the arrrangements and saw that they were efficiently carried out, I could mention several others, some of which occurred in places I had no opportunity of visiting, where as soon as the inhabitants bestirred themselves to follow the precepts laid down, the results were more or less as successful.

Wherefore I maintain that cholera is a preventible disease, and an easily preventible disease, which we understand and have the means of effectually stamping out, in contradistinction to plague and small-pox, concerning which we have only a comparatively imperfect knowledge, and I think we may look forward to a time when it will be as rare in India as Typhus is in England, a disease which not a hundred years ago was as prevalent there as cholera is here to day, but has almost entirely disappeared with the advance of sanitation.

Our efforts to check cholera so far have borne a certain amount of fruit as shown by the general decline in the figures for the last twenty years and with the ever increasing attention which is now being paid to sanitary matters we may confidently hope to see an infinitely greater improvement during the next bidecennial period.

The question of treatment strictly speaking does not come within our province, the business of the Sanitary Officers being to prevent people getting diseases and not to cure them. The first thing usually done on the appearance of an outbreak is to distribute medici es broadcast, most of the mofussil dispensaries keeping large quantities in stock, from which any one who applies for assistance is dosed. Doubtless a certain number of persons are relieved to some extent, but such a procedure does little or nothing towards combating the spread of the epidemic, and up to recent years apparently it represented the beginning, middle, and end of the efforts to stop it. Moreover of late in the Bombay Presidency the people have not taken kindly the medicines provided.

This however is not on all fours with the exhibition of acids as a prophylactic measure, bearing in mind Koch's sensational demonstration of the value of these drugs in that respect. I have always been in the habit of issuing 30m. of dilute hydrochloric acid in an ounce of water to clerks, servants and *patawallahs*, when touring in a cholera stricken district. They rather like the tart flavour and take it readily. So far I have never had a case amongst my establishment in camp although the disease has been raging in all the surrounding villages.

No dissertation on this subject would be complete without some reference to fairs and the part they play in spreading the malady. And we cannot but commend the action Government now proposes to take in holding searching official enquiries concerning them.

Every twelve years there is a very large religious gathering at Trimbak in the Nasik district, where lakhs of pilgrims congregate for a time, on the last occasion on which it was held, cholera was particularly virulent amongst them. When they dispersed in April we could trace the advance of the disease all down the main route they took to Poona, as they infected each village in turn on their way back to their homes. On reaching Poona they branched off to Satara and Sholapur, the former being particularly severely affected, and even introduced the complaint into Ratnagiri.

There is another pilgrim centre on the Bombay side, which for years used to be considered a menace to the health of the community so much so that the local Government voted 4¼ lakhs of rupees as a free gift to the municipality to provide them with a water supply which should be above suspicion.

This place, Pandharpur, is situated on the Bhima river, and is the seat of a very holy shrine (Vitoba), it has a population of 25,000 under ordinary circumstances, but during the Ashadi Fair which takes place annually at the beginning of the monsoon there are often as many as two hundred thousand persons collected there. Cholera used to make its appearance there every year as regular standing dish, and the pilgrims returning to their homes spread it all over the country. In 1906 a most violent epidemic raged there. It was commonly reported that there was one long and continuous procession of biers day and night to the cremation ground. There was not sufficient wood or dung-cake available to consume the bodies, and half burnt corpses were committed to the river wholesale. This state of affairs could not be allowed to continue and strenuous efforts were made to get the place into some semblance of a proper sanitary condition.

A special cholera hospital was built. Householders had to obtain a license to take in pilgrims as lodgers, the dwellings being previously inspected and passed as fit from a sanitary point of view to harbour them. They were directed to report any case of sickness occurring on their premises, under pain of having their license endorsed, under these circumstances we could safely insist on people being treated in the hospital.

A staff of medical subordinates were employed to examine every one entering the town, either by rail, road or river.

Stringent regulations were drawn up to prevent pilgrims obeying calls of nature in any place except those set apart for the purpose for which ample provision was made, and the usual stereotyped precautions were taken to prevent the drinking water being polluted, with the result that in 1909 when cholera was extremely prevalent all over the Deccan only about forty attacks occurred during the fair time and in the following seasons 1910 and 1911 not a single case appeared. This year the fair was stopped officially owing to the general prevalence of the disease throughout the country, railway booking to the station being prohibited for the time being, nevertheless some forty thousand people gathered there, but remained for the most part healthy. Pandharpur furnishes fewer cases now than any other towns of its size in the Presidency.

Proposed measures for dealing with cholera epidemics in the United Provinces.

During the years 1910 and 1911 the United Provinces were visited by severe cholera epidemics chiefly in rural areas and it was noted by the then Sanitary Commissioner, Major J. C. Robertson, I. M. S., that, in many districts, the epidemic appeared to run a course which was very little influenced by the measures taken to prevent its spread, until the district had been actually visited by the Sanitary Commissioner or a Deputy Sanitary Commissioner, and a proper organization initiated to combat the disease.

The methods adopted to cope with an epidemic by the district authorities are regulated by paras. 2022 to 2035, Manual of Government Orders, United Provinces. (See Appendix I). A perusal of these paras. makes it evident that the orders as they stand do not enjoin a sufficiently active campaign against the spread of the disease, especially at the commencement of the outbreak.

Para. 2022 says that Civil Officers should take measures for obtaining immediate information of the outbreak of cholera within their jurisdiction. I have not found, however, that the arrangements made in any district to obtain information in primary outbreaks are adequate. There are standing orders in most districts that the Chowkidar of a village in which cholera breaks out should immediately report the fact to the nearest *thana*. This, however, often appears to be disregarded and the report of an outbreak is usually made to the *thana* with his usual fortnightly or weekly mortality report. The *thana* then reports weekly to headquarters, who report to the District Magistrate. The latter informs the Civil Surgeon. The only agency the latter has at hand is that of the vaccinators. He recalls for cholera duty the vaccinators of the circle or circles from which the disease has been notified, serves them out with packets of permanganate of potash and orders them to proceed to the infected area and disinfect the wells.

As epidemics usually occur in the hot weather when the vaccinators are off duty, there is usually some difficulty in finding them so that more waste of time occurs. To cut a long story short, my experience is that no agency to disinfect wells is established as a rule until three weeks to a month have elapsed since the outbreak actually occurred and by that time as a rule the disease has spread over a considerable area and the vaccinators are quite unable to cope with it. As the mortality increases the Civil Surgeon usually applies for the services of one or two Sub-Assistant Surgeons who are given cholera medicines and are ordered to proceed to the infected area and direct the operations of the vaccinators and attend patients.

The Sanitary Commissioner receives the daily death returns and if the death-rate shows no signs of diminution, he deputes a Deputy Sanitary Commissioner to the district to advise the local authorities.

It has been my custom to advise the local authorities of any district which I visited during a severe epidemic to employ the revenue establishment to ensure the proper disinfection of all the wells in the epidemic area and to order all the tahsil officials to see that this was carried out in addition to their other duties and also in the case of severe epidemics to apply to Government for the services of one or more Tahsildars or Naib-Tahsildars for special cholera duty. I advise the purchase of large quantities of permanganate and the distribution of several weeks supply of this to the Patwaris in and adjacent to the infected area with orders to disinfect the wells twice weekly.

The tahsil authorities also had to see that all the possible breeding grounds of flies in the inhabited area were eradicated. As a result of the institution of these measures the death-rate always dropped rapidly. This year there was a severe outbreak of cholera in the Kheri district. I received orders from the Sanitary Commissioner while at Amritsar to proceed to Kheri on my return. I advised the measures above referred to and the advice was energetically carried out by the Deputy Commissioner, Mr. John Campbell. I arrived in Kheri on April 27th and left on the 29th. The following extract from a letter from Mr. Campbell will show the results :—

* * * * *

I have kept records in my office of the actual cholera mortality day by day, *i.e.* not the deaths reported day by day, but the actual deaths on any one day, the results work out as follows :—

20th-30th April	Average	65·5 per day.
1st week May	„	95·1 „ „
2nd „ „	„	22·8 „ „
3rd „ „	„	13·3 „ „

* * * * *

My *mahout* told me the other day that he had just come down from Nepal ; he said there were bodies lying about in the foot hills there by the hundred, unburied and unburnt ; no transport was procurable and there was no *bandobast* of any kind. His story is true I know. I have had any amount of independent verification of this.

* * * * *

Kheri is on the Nepal border and I quote the second extract to show the state of affairs within a few miles. The large diminution in the daily death-rate occurred at a season when a large increase in the death-rate occurred in previous years, so that the rapid diminution is entirely due to the efforts of the district authorities.

By the 1st week in June the death-rate was *nil* and since then only on one or two occasions have deaths been reported from Kheri district. These cases were imported and no spread occurred. In former years a rise in the cholera mortality was usual in August. This year the rise did not occur as the outbreaks were immediately stamped out.

The Sanitary Commissioner brought these excellent results and the methods used to obtain this to the notice of the Lieutenant-Governor and

the District Magistrate of Kheri also did so direct. The Lieutenant-Governor approved of the measures adopted and sanctioned the drawing up of a scheme for applying the same measures to certain districts in which cholera epidemics are prevalent and also the revision of certain of the paragraphs in the Manual of Government Orders with reference to cholera.

In order to ascertain how much permanganate of potash would be necessary to disinfect an average well, the Sanitary Commissioner requested me to carry out experiments as to the bactericidal effect of permanganate on cholera vibrios.

The results obtained are given in the two attached reports (Appendix II) to the Sanitary Commissioner.

I think that the results are most conclusive and that on an average one ounce of permanganate will be sufficient for an average sized well. After the completion of the experiment the proposed measures attached were drawn up for dealing with cholera in rural and in urban areas. A pamphlet on cholera for issue in affected villages was also compiled. The permanganate will be issued to all the officials concerned in the selected districts as soon as the proposals are sanctioned by Government so that the materials and the organization for dealing with an outbreak will be on the spot at the time of the outbreak.

With regard to the reporting it has been proposed that the police authorities should issue strict orders to all village Chowkidars to report primary cases of cholera to the *thana* immediately, having first informed the Patwaris. The Sub-Inspector at the *thana* will send these primary reports in green envelopes direct to the district headquarters, one to the District Magistrate and one to the Civil Surgeon, who will at once make arrangements for the deputation of vaccinators to the spot. The District Magistrate will then order the *tahsil* officials to supervise the work of the Patwaris and take steps to ensure the cleanliness of the infected villages as laid down in the attached proposals. (Appendix III.)

The revised proposals for outbreaks in towns are given in Appendix IV. The proposals as to immediate reporting should be incorporated in paragraph 2022. The measures to be taken on the occurrence of cholera in a town should be substituted for paragraph 2027.

The measures to be taken in rural areas should be substituted for paragraph 2032.

APPENDIX I.

Epidemic Diseases, such as Cholera.

Intimation of outbreak of cholera.

2022. Civil officers should take measures for obtaining immediate information of the outbreak of cholera within their jurisdiction, and should give immediate notice to (1) the military authorities, (2) the Magistrate of surrounding districts and to the Political Agents for neighbouring native States.

Note.—In cases of outbreaks at fairs, *see* paragraph 2070.

2023. Similarly all information received by the military authorities should be at once communicated by them to the chief civil authorities of the district.

2024. Civil officers should render every possible assistance to troops going into camp in consequence of an epidemic, especially in preventing any delay in procuring carriages, &c. The maintenance of carriage in anticipation of a possible demand is not expedient, but if district officers co-operate cordially with the military authorities, the rapid movement of the troops will be efficiently provided for. In carrying the measures into effect, expense may be incurred which the ordinary collection of carriage would not entail. Any such expenditure may be undertaken and will be a charge on the Military department.

Note.—Cholera camps should be ploughed up as soon as the troops quit them.

2025. The selection of encamping grounds to meet emergencies of this kind is to be made during the rains, when the suitability of any spot for a cholera camp can be easily tested.

2026. A civil officer should be appointed at each station to make the necessary inquiries as to the best sites; to select them in company of an officer of the Quarter Master-General's department, and to report the result to the Government through the Commissioner, after obtaining from the Officer Commanding the station any remarks that he may wish to offer.

Measures to be taken on the occurrence of cholera in town.

2027. A case of cholera under unhygienic conditions is a source of danger to its neighbours, and much can be done by early and suitable treatment of environment to prevent the extension of the malady. The following measures should be adopted forthwith on the occurrence of the first case; their general application, once the disease becomes epidemic, is almost impossible:—

(1) The floor, if *pakka*, of the room in which the sufferer is living should be washed down at once with a solution of perchloride of mercury (1 in 1,000) and to this four parts per 1,000 of hydrochloric acid should be added. The solution should be coloured blue. As soon as the patient dies or recovers, the floor should again be washed down with the same solution and the walls and ceiling treated in the same way. After two hours a disinfected floor should be washed down thoroughly with cold water. If the floor be *kachha* the earth should be removed to the depth

of four inches, the perchloride of mercury solution sprinkled on the ground, and four inches of fresh clean earth substituted.

(2) Upon the death or recovery of the patient, all the clothes worn by the sufferer should, if possible, be boiled or disinfected, and if likely to be spoilt by boiling or disinfection be exposed in the sun for eight hours. All rags and articles of no value which have come into contact with the patient should be burnt. The *charpai* upon which the sufferer has been lying, as also any other furniture with which he has come into contact, should be also washed down with the perchloride solution.

(3) Upon visiting the house in which a case of cholera has occurred the official should at once ascertain as to what latrine has been used by the sufferer. Usually it will be a latrine in the house. In this case the sweeper should be sent for, and it should be ascertained to what filth dépôt, or elsewhere the excreta have been removed. The receptacle and carts at such dépôt should at once be thoroughly washed out and disinfected with perchloride of mercury solution, as also the utensil in which the sweeper has removed the excreta from the house. The soil of the dépôt itself should also be dug up to the depth of four inches, disinfected, removed, and burnt.

Where a public latrine has been used, the entire latrine should be thoroughly washed down and treated with the perchloride of mercury solution.

(4) The latrine in the house needs particular attention. It should be dealt with whether the sufferer is stated to have used it or not. The whole of the interior of the structure, floor, wall, and ceiling should be well washed down with perchloride solution, and, in addition, if the floor be *kachha* the earth to the depth of four inches must be removed, and fresh, clean earth substituted. The *gamlas* should also be thoroughly disinfected, broken up, and buried. If the sufferer be dead, these measures will suffice, but if he be alive, the official should provide *gamlas*, four inches in depth, for the latrine and sick room and also furnish the house sweeper with a large bottle of the coloured acidulated perchloride of mercury solution (marked *zahr*) and instruct him and the family to disinfect each stool before removal. The urine should be similarly treated.

(5) Almost every private latrine (and nearly every dwelling has a latrine of some sort) has a drain, *pakka* or *kachha*, communicating with drain or cutting outside the house or hut. This drain must not only itself be disinfected, but also the public drain with which it communicates, for some distance from the house or hut. It can well be understood that it is highly dangerous for the washing from the latrine which has been used by a cholera case, to pass into the public surface drain in the crowded streets of a town or city. While the patient is still alive, and until death or recovery all these drains should be disinfected daily, as although the latrine may not be used by the sufferer, the residents of the house will probably throw foul matter into it.

(6) A very large proportion of houses (and even huts) in a town have a private well in the compound. The water in the well usually

being "bitter" (from nitrates, nitrites, and chlorides) is not often used for drinking purposes, except on an emergency, and the resident will usually say so; but it is used for washing plates and utensils and other purposes, and if it is contaminated or likely to become so .is distinctly dangerous. As the well mouth is usually flush, or nearly flush, with the ground, and as people bathe and wash clothes, &c., in its immediate vicinity, its pollution sooner or later is almost a matter of certainty. Where a case of cholera has occurred, the well should be treated with an ounce of permanganate of potash and sufficient hydrochloric acid to cause the water to slightly redden litmus paper. It should then be closed for a month, or if further cases occur in the same house, for a month after the recovery or death of the last case.

(7) A diffused and extensive outbreak should draw attention to the public water supply, and especially to the common use of a public well by those attacked; a more localized outbreak to the source of food supply; and an isolated case or cases to the possibility of the introduction of disease from without. The procedure in each case is clearly indicated. A filtered supply should be analysed and suitably dealt with; a public well closed or disinfected. A *Bania's* stock may have to be purchased and destroyed. But the last contingency will not often occur. In the majority of cases it is believed the origin of the disease will be found in the contamination of the water supply, very possibly by an arrival from an infected area.

(8) When the Assistant Surgeon or Sub-Assistant Surgeon in charge of a dispensary can spare the time, he should visit the houses of persons residing in the vicinity suffering from cholera, while the district vaccinator should be employed on the same duty in places beyond the reach of the dispensary officials.

Supply of medicines during outbreaks of cholera.

2028. (1) District officers should arrange for Civil Surgeons to be supplied with medicine trays containing sets of medicines to be sent out with Sub-Assistant Surgeons or vaccinators, when deputed to cholera-stricken parts of the country; the expense will be met from the local funds of the district, or, where there is a Municipal Committee from the Municipal Funds of the places which might require the services of the Sub-Assistant Surgeon.

(2) Cholera pills should be supplied free of charge, to postal officials in out-of-the-way places where there is no medical aid.

House inspection during outbreaks of cholera.

2029. While house-to-house visitation during epidemics of cholera is of the greatest importance, special hospitals are also necessary for the proper treatment of the homeless and destitute sick as well as for the isolation of cases of infectious disease. But any undue pressure used to compel the people to resort to such hospitals is likely to result in the concealment of cases of sickness to avoid removal from their homes.

Reporting of deaths from cholera.

2030. Whenever cholera prevails within the limits of any mortuary circle, the circle mortuary registrar shall submit a daily report of the deaths reported from cholera to the Magistrate in the prescribed form.

2031. The Magistrate will at the close of the day on which such reports have been received, or as soon after as possible, transmit in the same form a report to the Officer Commanding any cantonment within the limits of the district; or if there be no cantonment within those limits, he will report to the General Officer Commanding the Division. He will also forward reports in the same form to the District Mortuary Registrar, the Sanitary Commissioner, and the Commissioner simultaneously.

2032. In the event of a severe outbreak, the district mortuary registrar may, with the approval of the Magistrate, apply to the Inspector-General of Civil Hospitals for the services of one or more Sub-Assistant Surgeons to dispense medicines in the localities where the disease is most prevalent.

NOTE.—For the employment of vaccinators, &c., as distributors of medical relief during epidemics, the rules in the District Board Manual should be seen.

2033. In the case of a violent outbreak amongst the general population, or whenever the British troops in the district are attacked by cholera, the Magistrate shall report either fact direct to the Government.

2034. No exceptional measure having for its aim the arrest of the outbreak, such as the establishment of a local quarantine, prohibition of the sale of fruit or vegetables or of any article of food, shall be brought into force without the special permission of the Government.

2035. The Sanitary Commissioner shall watch the return of deaths from cholera and advise the Government when to call for a report as to the prevalence and cause of the epidemic.

APPENDIX II.

A report on the bactericidal effect of permanganate of potash on cholera vibrios.

1. *Procedure.*—The presence or absence of living cholera vibrios was tested by the cholera red reaction and by agglutination with high titre cholera serum. Cholera vibrios were seeded into flasks of 100 c.c. sterile water and Naini Tal lake water, ½ c.c. of a 24-hour growth in 10 c.c. of 1 per cent. peptone water was seeded into each without adding permanganate.

These were tested daily in order to ascertain how long cholera vibrios could live in a sterile water and a polluted water. The results of these experiments showed that living cholera vibrios could not be recovered from the polluted water after 24 hours but could still be recovered from sterile water in 7 days. I did not continue the tests of sterile water beyond 7 days.

A series of flasks of 100 c.c. each sterile water and lake water were then taken and ½ c.c. of a 24-hour growth of cholera vibrios in 10 c.c. of 1 per cent. peptone water was seeded into each. Immediately afterwards sufficient permanganate of potassium was added to make the solutions up to strengths of ½, ¼, and ⅛ of a grain per gallon.

Samples in a platinum loop were taken from the flasks at the end of ½ an hour, 1 hour, and 1½ hours and subcultured on agar slopes, and ½ c.c. in 10 c.c. of 1 per cent. peptone broth.

At the end of 24 hours' incubation the growths on agar were tested with high titre cholera serum for agglutination and the growths in peptone were tested for the cholera red reaction. The subculturing was done in order that the organisms, if any, surviving at the end of ½ an hour, 1 hour, and 1½ hours might grow and also because testing the original samples would give no differentiation between living and dead organisms as dead organisms of cholera also give the cholera red reaction and agglutinate with a high titre cholera serum.

The results of the tests are given below:—

Sample.	Quantity of permanganate of potash per gallon.	Time of contact with permanganate.	Cholera red reaction.	Agglutinations.
Lake.	⅛ Grain.	½ hour.	±	—
,,	¼ ,,	,,	±	—
,,	½ ,,	,,	—	—
,,	⅛ ,,	1 ,,	±	—
,,	¼ ,,	,,	—	—
,,	½ ,,	,,	—	—
,,	⅛ ,,	1½ hours.	±	—
,,	¼ ,,	,,	—	—
,,	½ ,,	,,	—	—
Sterile.	⅛ ,,	½ hour.	±	—
,,	¼ ,,	,,	±	—
,,	½ ,,	,,	±	—
,,	⅛ ,,	1 hour.	±	—
,,	¼ ,,	,,	±	—
,,	½ ,,	,,	±	—
,,	⅛ ,,	1½ hours.	±	—
,,	¼ ,,	,,	±	—
,,	½ ,,	,,	±	—

2. *Interpretation of results.*—The cholera red in all cases was extremely faint and is attributable to the amount of the sample added to the peptone water to make the subculture. This would contain dead bacilli which would give the faint pink tinge which was obtained. If the cholera vibrios subcultured had been alive there would have been a large increase of their numbers and consequently a strong cholera red reaction.

The agglutination tests were all negative showing that the permanganate had evidently killed the vibrios even in the greatest dilution used. The lake water in all cases gave growths on subculture but the organisms were not cholera. The sterile water also in some cases gave growths but these were contaminations. The chief organisms in Naini Tal lake water are fluorescent organisms and various members of the coli group.

3. *Conclusions.*—Permanganate in the strength used will kill cholera vibrios in ½ an hour in sterile water and in a polluted water. It therefore follows that any well water into which cholera vibrios have gained access should also be rendered innocuous. Taking 2,000 gallons as the average amount of water in a well, one ounce of permanganate should therefore be sufficient to render it innocuous.

A supplementary report on the bactericidal effect of permanganate of potash on cholera vibrios.

Using a similar procedure to that used in previous report I found that permanganate of potash in strength of 1/32 of a grain per gallon and 1/16 of a grain per gallon failed to kill the vibrios in one hour. I repeated the experiment with 1/8 of a grain per gallon and found that the vibrios were killed in one hour in spite of the fact that the amount of vibrios added was very great. I emulsified the whole of a 48-hour agar culture cholera at 37 c. c. in 5 c. c. of sterile water and added ½ c. c. of this to 100 c. c. of water. To this was added permanganate to make the strength of 1/8 of a grain per gallon.

In one hour 10 c. c. s. of this was added to 10 c. c. of 2% peptone broth making a 1% peptone broth medium. After incubating at 37 c. c. for twenty-four hours there was no growth. Using strengths of 1/32 and 1/16 of a grain per gallon of permanganate a growth appeared in the peptone broth.

This was sub-cultured on to agar slopes and the peptone broth tested with nitrate free pure sulphuric acid when the characteristic cholera red reaction was produced.

The agar sub-cultures were tested after 24 hours' incubation at 37 c. c. with high titre cholera serum. The organisms clumped readily confirming the result of the cholera red reaction.

APPENDIX III.

Measures for the immediate notification of cholera to the District Magistrate and Civil Surgeon.

I.—On the occurrence of any case or suspected case of cholera the Mukhia and Chowkidar of village or area in which the case occurred shall immediately inform the Patwari of the circle.

II.—On receipt of the news the Patwari shall immediately proceed to infected area or village to verify the report and shall on verification depute a Chowkidar to proceed at once to the nearest *thana*. The Thanadar shall arrange to send a duplicate message to District Magistrate and Civil Surgeon in green envelopes.

III.—The District Magistrate shall on receipt of the news give immediate notice to the Civil Surgeon as well as to the authorities as laid down in paras. 2022 and 2031 of the Manual of Government Orders.

IV.—Where the District Sanitary Officer is within the limits of the district the Civil Surgeon shall send information of the outbreak to that officer.

Measures to be taken by the Civil Surgeon.

I.—On receipt of the information of a cholera case the Civil Surgeon shall take immediate steps to depute vaccinators to proceed to the seat of the outbreak.

II.—The Civil Surgeon shall send with them a supply of cholera pills, potash permanganate, and medicines as laid down in Manual of Government Orders, para. 2028.

III.—The Civil Surgeon shall, when possible, visit the cholera infected village and advise the District Magistrate as to the staff required, in the event of an epidemic occurring. He should inspect the work of the staff placed on cholera duty.

IV.—No Civil Surgeon is allowed to proceed on leave if cholera has been reported within the previous 4 days.

Measures to be taken in rural areas to prevent the spread of cholera.

I.—The distribution of thirty-two one-ounce packets of permanganate of potash shall be made through the Tahsildar to each Patwari.

II.—Every Tahsildar shall in addition be supplied with a reserve stock of 10lb. of potash permanganate made up into 10 separate packages each containing 16 one-ounce packets of the drug for issue to Patwaris or Vaccinators and Sub-Assistant Surgeons on special cholera duty.

III.—Civil Surgeons should ascertain from the Tahsildars the number of Patwaris in the several tahsils of their districts and shall arrange to have an adequate stock of potassium permanganate in hand for distribution to Patwaris and Tahsildars.

IV.—Headquarter dispensaries will also keep a reserve stock of 10lb. of potash permanganate on which the Tahsildar may indent in case of emergency and on which any Sub-Assistant Surgeon or vaccinator placed on special cholera duty may also draw.

V.—On the issue of any of his reserve stock the Tahsildar or Medical Officer in charge of a dispensary shall immediately indent on the Civil Surgeon for the amount expended. The Civil Surgeon should replace amounts issued to vaccinators and Tahsildars by indent on D. Waldie and Company, Cawnpore, for renewals of stock.

VI.—Patwaris shall have orders issued to them to the effect that when a case of cholera occurs in their circles they must proceed at once to the infected area and disinfect the wells with permanganate of potash and redisinfect them every third day till the epidemic has ceased or till relieved by the vaccinator.

VII.—Tahsildars shall arrange for the removal of all refuse heaps for the purpose of improving the sanitary condition of the village and thus reducing the possible breeding grounds of flies.

VIII.—(1) District Magistrates should arrange for the distribution of pamphlets to all patwaris containing information as to the methods of avoiding infection from cholera.

The patwaris should be ordered to acquaint the villagers with the contents of the pamphlet.

(2) If the epidemic reaches the proportion of 20 deaths a day application should be made by the Magistrate to the Inspector-General of Civil Hospitals for the services of a Sub-Assistant Surgeon.

(3) In case of a severe epidemic within a fairly defined area, application should be made by the Magistrate to the Commissioner for sanction to depute a Tahsildar or Naib-Tahsildar on special cholera duty.

IX.—If the epidemic does not rapidly subside, the Civil Surgeon should apply to the Inspector-General of Civil Hospitals for the deputation to the infected area of the nearest travelling dispensary.

Method of disinfecting a well.

A *gharra*, bucket or *dol* should be filled with water from the well and the packet of permanganate poured into the water and well stirred with a stick. Three quarters of this dark red water should be poured into the well, taking care that the undissolved crystals of permanganate are not also emptied into the well. More water should be poured over the crystals and the process repeated until they are all dissolved, the bucket should then be lowered into the well and allowed to sink 2 feet and then pulled up sharply to mix the permanganate solution with the rest of the water in the well.

PAMPHLET ON CHOLERA.

Cholera seeds (germs) are carried from a cholera patient by water; or by flies; to the legs of which they adhere like wet *alsi* seeds cling to a stick.

Therefore—

I.—Use only water for drinking, or for washing out the mouth, which has been treated with *lal dawai* (permanganate of potash) or which has been boiled and kept in covered vessels to cool.

II.—Irrigation water is very dangerous as it may carry the cholera seeds from other villages in which cholera is present or even from a single case washing in it.

III.—When cholera is present, all food including milk should be well cooked and eaten while still warm, for cooking kills the cholera seeds in the same way as it kills grain seeds. Cold sweets or food bought in the bazar should be avoided; for flies carrying the cholera seeds may alight on cold food but usually avoid hot.

IV.—Eat no uncooked vegetables or fruit during a cholera outbreak.

V.—Drink nothing without at the same time taking food, because during digestion the contents of the stomach become acid and kill the cholera germ.

VI.—Take food before going out in the early morning.

VII.—Let no person suffering from cholera be near any food or drink that is meant for other people.

VIII.—Let no person prepare or eat food in the same room as a cholera patient.

IX.—Let nothing a cholera patient has used or that has been near a cholera case be put near food or water that is meant for other people.

X.—Everything that a cholera patient purges or vomits must fall into *gumlahs* half filled with dry earth or quicklime. All such earth, quicklime, and discharges, and at the termination of the case, the *gumlah*, must be buried in the ground 3 feet below the surface, but never near any well or spring or source of the water supply.

XI.—The bed, bedding, and clothes of a cholera patient, even if he has recovered, should be burnt.

APPENDIX IV.

Measures to be taken on the occurrence of cholera in dwellings and urban areas.

1. If cholera occurs in municipalities or notified areas and when the cases are few in number, a Municipal sweeper should be told off to each house in which there is a patient suffering from cholera. He should be supplied with Cyllin solution of the strength of 1-100 and with some earthenware *gamlahs* preferably glazed.

The sweeper should remove and disinfect the dejecta and vomit of the patient and should also disinfect the floor, *charpai* and bed clothes if polluted and also the house latrine.

2. Where a Municipal sweeper is not available the following steps must be taken :—

The floor of the room in which the sufferer is living if *pakka* should be washed down with 1 in 100 Cyllin solution every time it is soiled by faeces and vomits. As soon as the patient dies or recovers, the floors and walls and beds should be washed down with the same solution.

If the floor be *kachcha* the earth should be removed to a depth of 4 inches—quicklime sprinkled on the ground and 4 inches of fresh, clean earth substituted. If the earth is not removed the floor should be thickly covered with quicklime or covered with grass which should be set alight.

3. Upon the death or recovery of the patient, all the clothes which had been worn by him should, if possible, be boiled or disinfected, and if likely to be spoilt by boiling or disinfection, be exposed in the sun for 8 hours. All rags and articles of no value which have come into contact with the patient should be burnt. The *charpai* upon which the patient has been lying and also any other furniture with which he has come into contact should be also washed down with Cyllin solution.

4. Upon visiting the house in which a case of cholera has occurred the officials will at once ascertain what latrine has been used by the patient. Usually it will be a latrine in the house. In this case the sweeper should be sent for and it should be ascertained to what filth depôt, or elsewhere, the excreta have been removed. The receptacles and carts at such dépôt should at once be thoroughly washed out and disinfected with Cyllin and retarred.

The soil of the dépôt itself should also be dug up to a depth of 4 inches, disinfected, removed, and burnt or treated as in paragraph 2.

Where a public latrine has been used, the entire latrine should be thoroughly washed down and treated with Cyllin, and retarred if an iron latrine.

5. The latrine in the house needs particular attention. It should be dealt with whether the patient is stated to have used it or not. The whole of the interior of the structure, floors, walls, and ceiling should be well washed down with Cyllin solution, and, in addition, if the floor be

kachcha the earth to the depth of four inches must be removed, and fresh, clean earth substituted. The *gamlahs* should also be thoroughly disinfected, broken up, and buried. If the patient has died, these measures will suffice, but if he be still alive, the officials should provide *gamlahs* (preferably glazed) 4 inches in depth, for the latrine and sick room and also furnish the house sweeper with Cyllin solution and instruct him and the family to disinfect each stool before removal.

The *kachcha* floor should be sprinkled thickly with quicklime or treated as in paragraph 2.

6. Almost every private latrine (and nearly every dwelling has a latrine of some sort), has a drain, *pakka* or *kachcha*, communicating with a drain or cutting outside house or hut. It can well be understood that it is highly dangerous for the washings from the latrine which has been used by a cholera case, to pass into the public surface drains in the crowded streets of a town or city. While the patient is still alive, and until death or recovery, all these drains should be disinfected daily, as although the latrine may not be used by the patient, the residents of the house will probably throw foul matter into it.

7. A very large proportion of houses (and even huts) in a town have a private well in the compound. The water in the well usually being "bitter" (from nitrates, nitrites, and chlorides) is not often used for drinking purposes, except on an emergency, and the resident will usually say so: but it is used for washing plates and utensils and other purposes, and if it is contaminated or likely to become so is distinctly dangerous. As the well mouth is usually flush, or nearly flush, with the ground, and as people bathe and wash clothes, &c., in its immediate vicinity, its pollution sooner or later is almost a matter of certainty. Where a case of cholera has occurred, the well should be treated with an ounce of permanganate of potash and sufficient hydrochloric acid to cause the water to slightly redden litmus paper. It should then be closed for a month, or if further cases occur in the same house, for a month after the recovery or death of the last case.

8. A diffused and extensive outbreak should draw attention to the public water supply, and especially to the common use of a public well by those attacked; a more localized outbreak to the source of food supply; and an isolated case or cases to the possibility of the introduction of disease from without. The procedure in each case is clearly indicated. A filtered supply should be analyzed and suitably dealt with; a public well closed or disinfected. A *Bania's* stock may have to be purchased and destroyed. But the last contingency will not often occur. In the majority of cases it is believed the origin of the disease will be found in the contamination of the water supply; very possibly by an arrival from an infected area.

9. Where possible and where supervision can be exercised the mouths of all the wells in the infected village or town should be temporarily closed by boards and sods. One or two of the best wells should alone be kept open. These wells should be permanganated and for them

Kahar water-drawers should be appointed. No person except the Kahars appointed for this purpose is permitted to draw water from these wells. The Kahars should be furnished with a new rope or one that has been well soaked in permanganate. At each end of the rope should be affixed an iron bucket or a kerosine oil tin. These tins and *dols* should never be removed from the well during the outbreak. The Kahars furnish the villagers with water by pouring it into a hollow bamboo or tin *parnalla* below one end of which the villager presents the water vessel which requires filling. This method is commonly known as the *piau* system. The services of the Kahars should be retained for six days after the occurrence of the last cholera case.

ALL-INDIA SANITARY CONFERENCE—MADRAS—NOVEMBER 1912.

THE BACTERIOLOGY OF CHOLERA AND ITS RELATION TO THE SPREAD OF THE DISEASE FROM THE POINT OF VIEW OF THE HEALTH OFFICER.

BY

Dr. J. A. Turner, M.B., D.P.H., Executive Health Officer, Bombay Municipality.

The following notes may be of interest in the very difficult question of the relation of the Cholera Vibrio of Koch to the spread of Cholera.

These experiments were carried out during the recent outbreak of Cholera in Bombay, June—August, 1912.

From these investigations, I draw the following conclusions :—

(1) That the Comma Vibrio is present in the excreta of persons dying or suffering from Cholera.

(2) That the Vibrio persists until some time after the patient is convalescent but not indefinitely.

(3) That in times of Cholera epidemic the Vibrio is found in waters, wells, tanks, vessels, etc., but that it does not retain its morphological and cultural characteristic or its virulence for any length of time.

(4) That an outbreak of Cholera depends on:—

1. The strength and quantity of the dose of the virus received.
2. The condition of the infected material which gains access to the intestine.
3. The physical conditions of the patient and his power of resistance.
4. Certain climatic conditions.
5. Sanitary surroundings of the people, water, food, drainage and refuse disposal and the habits of the people.

(5) That when the Vibrio leaves the infected person it loses its virulence in direct ratio to the time and conditions to which it is subjected.

Bacteriology.

Bacteriology. The bacteriology of Cholera has provided the subject for an enormous amount of work. Koch, who discovered the Comma bacillus Vibrio, visited India in 1883 and was followed by Klein and Gibbes in 1885 and later by Haffkine.

There is an enormous field for further scientific research in the bacteriology and epidemiology of Cholera.

With the improved technique of the laboratory and the opportunities now given for research work in tropical diseases, the investigations into the bacteriology and epidemiology of Cholera should be considered of the first importance.

It is unnecessary to repeat the investigations of eminent observers and the enormous amount of work done in the bacteriology of Cholera which have

brought us to our present knowledge. It is accepted, rightly or wrongly, that the Vibrio called the Comma Vibrio of Koch is associated with Cholera; that the organism exists in the stools of Cholera patients, that it can be isolated and can exist in the water.

That the stools of Cholera patients, either directly from the body or from the clothes and discharges, can infect water and food and milk and thereby convey the disease.

During a severe outbreak of Cholera in Bombay from our investigations by daily visits of inspection to the infected houses, streets, wells and tanks in B Ward and from bacteriological examination, there is sufficient evidence to assume that the water had been polluted directly or indirectly from Cholera cases.

A great deal of work has been done with the Cholera Vibrio and the laboratory methods and technique are delicate and uncertain.

In order to ascertain the probable cause of an outbreak, in addition to the epidemiological investigations, we examined many samples of water from wells, tanks, open vessels and tap water as well as *post mortem* examination of intestines and the fæces of patients dying of the disease.

During January to August, 114 samples of Municipal waters were examined in the laboratory; 257 samples of other waters, 16 of milk and food stuffs and 18 of excreta of Cholera patients. These examinations were conducted until September and the fæces of 62 patients examined.

During my visits of inspection my attention was drawn to the following:—

(1) To provide water for the people large earthen vessels were placed in the streets.

They were filled with water by Bhistis from tanks, stand-pipes, and wells.

(2) That during certain hours of the day and night the Municipal water-supply was shut off owing to the scarcity of supply and that Bhistis were providing it to the houses.

(3) That stand-pipes in the City are used as bathing places and for washing clothes and utensils.

(4) That Bhistis filled their mushaks at these stand-pipes immediately before, after, or, if possible, at the time people were washing their bodies and clothes.

(5) The large reservior at Pydhoni adjoining the Police Station was a favourite place for this purpose, and although there is a notice pointing out that these stand-pipes were not meant for washing bodies and clothes and although it adjoins the Police Station, dozens of people are to be seen daily fighting for water and washing their clothes.

(6) This was pointed out to the Police, who temporarily stopped it.

(7) Bhistis when they cannot get a supply of water from stand-pipes get the water from tanks, wells, at mosques and other places, and carry the water to private houses, where it is stored in various kinds of tubs, earthenware pots, etc.

The possibility of conveying infection through water is apparent.

The Bhistis' mushaks during the outbreak were washed with lime and permanganate solutions.

Now supposing that water is contaminated with material from an infected house, person or clothes, it is not unreasonable to assume the probability of the disease being spread in this way.

I therefore took samples from open vessels in the streets and houses, tanks and wells in the mosques.

It was found, from the result of the analyses of the many samples that were sent, that the majority of the waters were contaminated with fæcal matter, and

in some instances an organism having cultural characteristics of the Cholera Vibrio.

Although a Vibrio was present in many samples, it was not until we examined a sample from a well in a mosque in Kazi Syed Street that we arrived at what appeared to be the real Cholera Vibrio.

The well is inside the mosque and adjoining it, a tank and a place where dead bodies are washed in the mosque. Three deaths from Cholera occurred here and many dead bodies were washed in a place close to the well and tank. Outside the mosques are galvanised vessels filled with water for the use of the passers-by.

As has been pointed out, to obtain a pure culture of the Cholera Vibrio in a well or tank water is not easy, specially in India, where many other vibrio exist.

Water is much fouled with organic matter, and the presence of other bacteria to a certain extent smother the Cholera Vibrio.

Captain Gloster, I.M.S., who was deputed to study the outbreak of Cholera on behalf of the Scientific Research Committee, also took a sample of the water, but did not pursue the investigation on account of innumerable bacteria present, but said that fæcal matter was present.

Captain Gloster, however, made further research into this particular organism sent to him by us, and he submitted it to the many tests which must be positive, or the vibrio, according to some observers, cannot be accepted as a true Cholera Vibrio.

Captain Gloster did not find this organism agglutinating to Anti-Cholera serum nor to give a positive reaction with Pfeiffer's test.

The bacteriological tests which were used in the Municipal Laboratory are as follows :—

(1) 90 c. c. of the water to be examined was put in a narrow mouthed flask with 10 c. c. of an alkaline concentrated peptone solution and incubated for 24 hours.

(2) This was then examined microscopically and after staining with carbol fuchine and if vibrios were present—

(3) a loop full of the peptone solution was inoculated into a sterile broth culture medium.

(4) A sub-culture was immediately made in agar slant tube.

(5) Further dilutions were made in another broth tube.

(6) A further sub-culture was made into slant agar tubes and incubated for 24 hours, and then examined microscopically, as in No. 2.

(7) Colonies from the agar were then further sub-cultured on agar and subjected to other tests.

Another method—48 hours' peptone cultures were plated out on agar petri dishes and incubated for 24 hours and the colonies were examined.

Pure cultures were then examined as follows :—

Microscopically, stained as before, motility, Cholera red re-action, Nitroso indol, Ehrlich Rosindol reaction—staining for flagella—agglutination and Pfeiffer's test.

All the tests mentioned above were applied to the Vibro, isolated from this well water, in the Municipal Laboratory and found to be positive with the exception of the Pfeiffer test.

This was undertaken by Captain Gloster at the Parel Laboratory and was found to be negative, nor did he get agglutination.

Now if there is any organism that varies in its cultural, morphological and pathogenic properties, it is the Cholera Vibrio, and from the latest investigations,

with the most modern bacteriological technique, it is stated that "the power of Anti-Cholera serum to agglutinate the Vibrio is very variable."

"That because either agglutination or Pfeiffer's test is negative, it does not follow that in the presence of an epidemic a Vibrio not responding to these tests should not be considered to be a Cholera Vibrio for all practical purposes."

Under the circumstances I consider that from our investigations it is proved that this well water was infected with material from a Cholera patient, and that it was possible many other wells and tanks and water vessels were infected in a similar way.

This is as far as any observers have ever got in the bacteriology of the disease, and until we know more we must assume that Cholera is spread in this way.

I maintain for the practical sanitarian this is enough to work upon.

Given an outbreak of Cholera, whether imported or indigenous, any sample of water, from which a pure culture of the Comma Vibrio is obtained, is sufficient evidence for active preventive measures.

The Municipal Laboratory cannot, for obvious reasons, undertake that high standard of research which this question deserves.

I am strongly of opinion that the Cholera Vibrio, after it leaves the human body, undergoes such rapid modifications in water, milk, food, soil, or even on the soiled clothes and discharge of the suffering person, as to render it almost unrecognisable.

The organism, fortunately for the human race, is very faintly resistant to external influences; and it is for this reason, that when an outbreak occurs, prompt sanitary measures can control it.

How far, and how long, a Cholera Vibrio can retain its virulence, externally to the human body, and how far the other organisms in water affect it, are still subjects for further investigation.

How the attenuated organism, found in water, can again regain its virulence when entering the body of its former host, is what we want to know.

Until then the practical sanitarian must consider all water containing a Comma Vibrio, in the presence of a Cholera outbreak, a danger, which by all the means in his power, should be attacked.

Without entering into the bacteriology of the innumerable Cholera-like vibrios found in water and fæces, the presence of a Comma Vibrio in water during a Cholera epidemic is evidence of contamination from a case of Cholera. It is, as I have already pointed out, held that infected water is always the cause of the spread of Cholera, and that personal contact has little to do with the spread of the disease.

Personal element in the spread of Cholera.

It is impossible to analyse the evidence collected during the outbreak in Bombay without coming to the conclusion that the personal element has a great deal to do with the spread of Cholera in India. Without labouring too much on the habits and customs of the people, I am strongly of opinion that the spread of Cholera is greatly due to direct infection from person to person by means of the discharges, contaminated hands, food, vessels, milk and water and flies.

The filthy privy baskets, over-flowing into open drains and thus soaking into sub-soil or storm-water drains, the position of the water sewer pipes and the common washing places are a constant danger at any time, but, in the presence of Cholera, must contribute to a large extent to the circumstances favouring the spread of the disease.

For example, on visiting a person suffering from the disease, he or she will be surrounded with relatives and friends handling the patient. Many will be found occupying the same room, sleeping on the floor; they all use the same privy and washing place, eat from the same vessels; the water is stored in wooden, iron, earthenware, or other vessels, in the living room, cook-room or

nahani, for days together. The friends and relatives will wash the dead body and take it to the burial ground or burning ghat and afterwards wash in the nearest tank or bathing place—without any disinfectant precautions.

Any one familiar with the homes of the poor can easily understand how Cholera is thus spread.

The following are some instances:—

A man, Hindu, living in a well-built chawl, the servants' quarters of a large bungalow on Malabar Hill, occupied a room by himself; he had his meals with a man and woman in an adjoining room. No case of Cholera had occurred in the neighbourhood. He visited the Bazar one day, came home and took ill with vomitting and diarrhœa; he was nursed, fed and attended to by a woman in the adjoining room. This man was removed to Hospital that evening in a collapsed condition and died two hours afterwards.

Next day, the woman, who had attended him, took ill with diarrhœa and vomitting, and after some difficulty was persuaded to go to Hospital, and she recovered.

House No. 2, C. Street, three deaths occurred on the ground and 1st floor occupied by the same family, well-to-do Mahomedans.

The first case occurred on the 12th, the second on the 13th, the third on the 14th. The house is situated back to back, separated by a narrow sweeper's passage, or gully, from a house in B. Street, where seven deaths occurred, four on the 11th.

From house No. 25, friends and relatives visited house No. 2, where the three cases were suffering; three deaths occurred at house No. 25, on the 14th, 19th and 19th.

From house No. 25, a suffering case was taken to house No. 1-3, M. Street, and died there.

At house No. 25, a lady came to visit and remained there some days, took ill, was taken to No. 207. A. O. Street in C Ward, and died there.

A number of similar instances are given in statements referred to.

CONCLUSIONS.

What we want to know is how far a person convalescent from the disease, or suffering from it in a mild unrecognised form, can be a danger to the community as a "carrier."

May such a case, unknowingly, convey the infection to *nahanis*, privies, washing and drinking vessels, or the food supply or even a public water-supply, and thus start an outbreak in the neighbourhood.

May and June were unusually hot months. The Municipal water-supply had been curtailed in B Ward.

On the 10th of June and some days previous, large collections of Mahomedans congregated in Mandvi, Umerkhadi and Chuckla.

Water had to be provided for these large crowds.

Given then a few cases of Cholera in these Sections, it is possible that facilities were offered to the pollution of the temporary water-supply provided for the people which was infected and thus caused the sudden rise in the incidence of the disease.

As soon as the temporary supply of water was abolished, tanks and wells disinfected and cleaned, the disease began to disappear.

In the bacteriological report it will be seen that the Cholera Vibrio was only found in one tank, but as many of the others had already been cleaned or disinfected, it is not to be expected that this very volatile organism would be found there.

This is nothing new in the epidemiology of Cholera, as witness the Fairs held in India.

Large crowds of people assemble, Cholera breaks out, and is only checked when the Fair is over, or the proper sanitary precautions have been taken to prevent the pollution of the water-supply, and other sanitary precautions observed with regard to food and cleanliness of the camps, and destruction of all infected material.

Many persons leaving the Fairs perfectly well, are attacked or die on their way home or recover and return to their homes convalescent, thus becoming a focus to spread the disease elsewhere.

Facilities of transit are so many that the disease may be carried hundreds of miles in a few hours.

Bombay City is a very small unit of the Indian Empire or even of the Bombay Presidency, but it is the haven of refuge for the destitute as well as the prosperous in times of famine and sickness in the neighbouring districts, and thus pays the penalty of its position.

The difficulties of obtaining information from the friends and relatives of infected people, and the passive resistance to any sanitary measures, make it extremely difficult to trace cases of infectious disease or to adopt measures for their control.

The indifference and carelessness on the part of the poor people to accept any advice adds to the difficulties of control, while the ignorance of any measures of personal hygiene and the rooted objection to any innovation in their domestic arrangements, form obstacles difficult to overcome.

The bateriological examinations were carried out by the Municipal analyst, Dr. L. Joshi, and his assistant, Mr. Pansari.

The following routine method was used in the examination of the samples for Cholera :—

I.—*For samples of waters*—

90 c. c. of the water is transferred to a narrow-mouthed sterile flask containing 10 c. c. of concentrated peptone solution. This is placed in the incubator at 37° C. for 24 hours.

II.—Smears are now made and examined microscopically after staining with 1-10 Carbofuchsim. If the smears show any Cholera-like organisms then one of the following methods are followed in isolating the pure culture of Cholera Vibrio.

III.—*Method A.*—

(*a*) One loopful is removed from Flask (No. 1) containing 24 hours culture on peptone and put in a broth tube. This is labelled No. 2.

(*b*) A sub-culture is immediately made on slant agar (2*a*).

(*c*) Further dilution is now made by taking a loopful from (2) and putting it in a broth tube labelled (3).

(*d*) An immediate sub-culture is made from (3) into slant agar 3(*a*). The tubes are then incubated at 37° C. for 24 hours.

(*e*) At the end of 24 hours several suspicious-looking colonies are selected from 2*a* and 3*a* and examined in the usual way microscopically.

(*f*) Such isolated colonies are again sub-cultured on agar for 24 hours, after which they are subjected to other tests for Cholera Vibrio (*q. v.*)

Method B. for isolation of Cholera.—

In this method 48 hours' old peptone cultures are plated on petri dishes and incubated at 37° C. for 24 hours. Several colonies are picked up from these and examined in the usual way.

IV.—*The pure sub-culture obtained by method A are now examined as follows:—*

(1) Morphology.

(2) Motility.

(3) Cholera Red Reaction—

(*a*) The nitroso-indol. Reaction with sulphuric acid (this is tried in 24 and 48 hours).

(*b*) Ehrlich's Rosindol Reaction with Paradi-methy-lami-dobeyal-dehyde. This is also tried in 24 and 48 hours.

V.—*Staining for Flagella—*

Stephen's stain is used on a fresh sub-culture (under 24 hours).

VI.—*Agglutination test—*

This is tried with serum usually obtained from the Bombay Bacteriological Laboratory, Parel.

VII.—Method for examining samples of fæces from Cholera patients.

These were either passed through peptone water and isolated on slant agar or diluted through broth and immediately inoculated on slant agar. The rest of the method was the same as in the case of water.

The method of examining samples other than those of waters or of fæces does not differ materially from method No. 1 (*q. v.*) excepting that smaller amount of peptone water is used up.

Pfeiffer's Reaction and Growth on Sugar Media.

These were in most cases examined for us by Captain Gloster, I.M.S., at the Parel Laboratory, who was supplied with pure sub-cultures of Cholera Vibrio isolated by us from waters, etc.

In the present investigation the main difficulty was felt in the isolation of a pure culture of Cholera-like bacilli from all the other organisms present in the water. This is the main difficulty usually experienced by most bacteriologists. After trying various methods we were fortunate enough to isolate a pure culture by method A. As described above this method is simple, quick, and there is no risk of external contamination.

Method B, although used by many bacteriologists, was not found so satisfactory. Besides in method B there are many chances of external contamination during the manipulations.

The isolated organisms were typically comma-shaped with marked curve, actively motile, with one terminal flagellum, gave the "Cholera Red" reaction within 24 and 48 hours, showed complete or partial agglutination, gave acid but no gas in sugar media.

The agglutination test was not positive in all cases and this may be perhaps due to the serum not being homologous to the organisms isolated by us.

Out of the 370 samples of waters examined for Cholera Vibrio, it was detected only in two samples of well waters—(1) well at No. 34-36, Mount Road, and (2) well at Bohra's Mosque, No. 10, Cazi Sayyad Street—with positive results both microscopically and culturally. Besides comma-like bacilli were seen microscopically in 16 samples with negative results for cultural tests as follows:—

5 well waters; 6 tap waters; 3 tank waters; 1 water from earthen pot, and one not classed.

Cholera Carriers.

Extract from a report prepared by Dr. Crendiropoulo, Director of the Bacteriological Laboratory of Chatby, Alexandria, published by the Conseil Sanitaire, Maritime, and Quarantenaire of Egypt, upon an examination of the stools of travellers coming from countries infected with cholera. The investigation was started owing to the following incident. On August 16th, 1911, a Belgian steamer arrived at Alexandria from Smyrna, where cholera was present.

On the fifth day of the period of quarantine a little girl 5 years of age died with symptoms suggestive of cholera. At the necropsy the characteristic lesions of this disease were not found, but bacteriological examination revealed the presence of abundant vibrios, and cultures made from them were powerfully agglutinated by cholera serum. Examination of the stools of all the other persons on board the steamer showed that one woman, a servant in the family to which the dead child belonged, was a carrier of an agglutinating vibrio. Other incidents of similar nature occurring within a few days showed the danger these carriers represented for Egypt, and the Conseil Quarantenaire, on the proposition of Dr. Armand Ruffer, authorized the quarantine authorities to carry out bacteriological examinations as part of their visit in all cases where they considered it necessary. In consequence, between the dates of August 17th 1911 and January 31st 1912, the passengers and crews of 297 vessels were submitted to an examination of the stools as part of the routine examination for quarantine. The total number of stools examined was 34,461, and of these 14,553 were obtained from the crews and 19,908, from passengers, mostly from those of the poor class, although in some instances every person on the boat was examined. Vibrios were found in 63 cases, and of these 23 possessed agglutinating properties with cholera serum, while 40 did not. Of the 23 agglutinating vibrios only 2 came from members of the crews, while of the 40 non-agglutinating, 12 were found in members of the crews. The percentage of agglutinating vibrios was therefore about 0·07, but it was found that in the 44 days from August 17th to September 30th, during which the epidemic was at its height, there were ten times more carriers than during the remainder of the period. In general, Dr. Crendiropoulo finds that agglutinating vibrios are only found in travellers from infected countries and only when an epidemic is at its height. During the decline of an epidemic carriers become extremely rare. It is interesting to note that the greater number of carriers were found in passengers and that the crews were but rarely affected. All the carriers were kept in quarantine until the disappearance of their vibrios, which took place in most cases within five days, though in one case they persisted for eight days. Dr. Crendiropoulo is careful to point out, however, that the limit of five to eight days must not be taken as the ordinary time of persistence of the vibrios, since they had probably been present for some time before they were discovered. In regard to the non-agglutinating vibrios, their frequency is found to be in inverse proportion to that of the agglutinating. They become more abundant as the carriers of true cholera vibrios become fewer, but both of them cease altogether when the epidemic comes to an end. Dr. Crendiropoulo gives an interesting and exhaustive account of the bacteriological characters of the vibrios obtained, including their virulence for the pigeon, liquefaction of gelatin, coagulation of milk, the production of haemolysis and the indol reaction, and he concludes that none of them can be relied upon as a criterion for the diagnosis of the cholera vibrios. Even the fixation of complement and the agglutination reactions are not free from doubt. As a result of his observations he suggests that every carrier of vibrios, whether agglutinating or not, who comes from a place where the disease is epidemic, should be held as suspect.

Summary of Table II.

Experiments to show that cholera bacilli could be isolated from artificially infected tap-water up till the 4th day only.

Series I.

A flask containing 1000 CC of tap-water was infected with a strain of Cholera Bacilli isolated from patient No. 1350.

90 CC of the water were taken from the flask on the 2nd day, 3rd day, and 4th day, and in each case a pure culture of Cholera Bacilli could be isolated.

On the 5th and 6th day no Cholera Bacilli could be separated.

Series II.

The same experiments were tried with Cholera Bacilli isolated from patient No. 1351.

The same results as above were obtained, *viz.*, pure cultures of Cholera Bacilli could be obtained till the 4th day, but on the 5th and 6th no Cholera Vibrios could be isolated.

The usual methods were adopted in isolation of the Cholera Bacilli.

TABLE I.

The vitality of Cholera vibrios isolated from the fæces of cholera patients (Arthur Road Hospital) in artificially infected samples of Laboratory tap water.

Date.	Experiment.	Volume of Laboratory tap water.	Age of cholera culture used.	Strain of cholera used.		Results as regards isolation of cholera vibrio from the infected Laboratory tap water.	
				Serial No.	Patient's No.		
21st August 1912	1	90 C.C. ...	24 hours ...	1959 ...	1289 ... (convalescent)	Positive after 24 hours.	
Ditto	2	„ „ ...	„ „ ...	„ ...	„ ...	Negative after 8 days.	
Ditto	3	„ „	„ „ ...	1986 ...	1299 ... (convalescent)	Positive after 24 hours.	* In this case a culture resembling cholera by physical appearance on agar was obtained but to the further tests it was found negative. Microscopic, somewhat curved but thick bacillus.
Ditto	4	„ „ ...	„ „ ...	„ ...	„ ...	Negative after 8 days.	
Ditto	5	„ „ ...	3 days ...	2080 ...	1350 ...	Negative after 2 days.	
Ditto	6	„ „ ...	„ „ ...	„ ...	„ ...	Negative after 4 days.	
Ditto	7	„ „ ...	„ „ ...	2081 ...	1351 ...	Negative after 2 days.*	
Ditto	8	„ „ ...	„ „ ...	„ ...	„ ...	Negative after 2 days.	

TABLE II.

The vitality of Cholera vibrios isolated from the fæces of cholera patients (Arthur Road Hospital) in artificially infected samples of Laboratory tap water.

Date.	Experiment.	Volume of Laboratory tap water.	Age of cholera culture used.	Strain of cholera used.		Results as to presence of comma bacilli microscopically.	
				Serial No.	Patient's No.		
23rd September 1912	1	90 C. C.	24 hours	2080	1350	Positive after 48 hours.	
" " "	2	" "	" "	2081	1351	" " 48 "	
24th " "	3	" "	" "	2080	1350	" " 72 "	A pure culture obtained.
" " "	4	" "	" "	2081	1351	" " " "	A pure culture obtained.
25th " "	5	" "	" "	2080	1350	" " 96 "	
" " "	6	" "	" "	2081	1351	" " " "	
26th " "	7	" "	" "	2080	1350	Negative after 5 days.	
" " "	8	" "	" "	2081	1351	" " 5 "	
27th " "	9	" "	" "	2080	1350	" " 6 "	
" " "	10	" "	" "	2081	1351	" " " "	

TABLE A.

The vitality of cholera vibrios isolated from the fæces of cholera patients (Arthur Road Hospital) in artificially infected samples of sterile tap water.

Date.	Volume of sterile tap water.	Age of the cholera culture used.	Experiment.	Strain of cholera used.		Results as regards isolation.
				Serial No.	Patient's No.	
21st August 1912	20 C. C.	24 hours	1	1959	1289	Positive up to the 45th day.
" " "	" "	" "	2	1986	1299	" " " " "
31st August 1912	" "	3 days	3	2080	1350	" " " 35th day.
" " "	" "	" "	4	2081	1351	" " " " "

Effect of the Sun's rays.

On Cholera Vibrios. Strain 2080.

26th September 1912. Experiment I. One loopful cholera growth on agar added to sterile tap water in a test tube. The depth of the water was 3 inches. The tube was exposed to the sun for 3 hours, from 11-15 A.M. to 2-15 P.M. The temperature of the water was ranging from 43° to 45°C.

Result :—The Cholera Vibrios were killed.

28th September 1912. Experiment II. The same process was adopted. The tube was exposed for 2 hours, from 10 A.M. to 12 A.M. The temperature was ranging from 39° to 40°C.

Result :—The Cholera Vibrios were killed.

1st October 1912. The same process. The tube was exposed for 1½ hours, from 12-20 to 1-50 P.M. Temperature 39° to 40°C.

Result :—The Cholera Vibrios were killed.

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Bacteriological Analysis of water for cholera.

Serial No.	Source as per label.	Received on	Remarks.
1	Well water for Cholera, 36 Mount Road; well treated with Pot. Permgte, on 5th July 1912; water used by H. H. Aya-khan's and many Khoja families in Bom-bay; received from Deputy Health Officer E. Ward.	27-7-12 at 11-50 a.m.	Comma bacilli present; isolated pure colony from Agar. Indol and Rosindal positive both for 24 and 48 hours; Agglutination 1 : 40 negative. Flagellum; one Terminal present.
2	Tank water for Cholera obtained from Bara Masijd at Dhabu Street, No. 76 sent by District Registrar No. 5 District.	30-7-12 at 11-15 a.m.	A few comma shaped bacilli seen microscopically but cultural tests for Cholera negative.
3	Storage Tank water for Cholera obtained from Edward Sassoon Mill, Fergusson Road, through Deputy Health Officer G. Ward.	31-7-12 at 12 Noon.	Comma bacilli present microscopically : cultural tests for Cholera negative.
4	Water from "Goohas" used for drinking purposes from Maneckji Petit Mill (through Deputy Health Officer E.).	3-8-12 at 9·55 a.m.	Negative for Cholera.
5	Storage Tank water (drinking) obtained from Maneckji Petit Mill (through Deputy Health Officer E.).	3-8-12 at 9·55 a.m.	Both negative for Cholera.
6	Tapwater A. M. P. Mill (through Deputy Health Officer E.).		
7	Tapwater Maneckji Petit Mill B. ...	...	Comma present; Indol present; Flagellum present; Agglutination 1 : 40 Negative.
8	Well water for Cholera obtained from New Chall No. II, New Parbadevi Road (through Deputy Health Officer G.).	3-8-12 at 11-30 a.m.	A few commas microscopically but cultural tests for Cholera negative.
9	Tapwater for Cholera marked A. Maneckji Petit Mill handed in by Dr. Sorab.	5-8-12 at 4 p.m.	No commas detected.
10	Tapwater for Cholera marked B. Maneckji Petit Mill handed in by Dr. Sorab.	...	Commas present; Indol positive both for 24 and 48 hours: Motility pre-sent: Flagellum were Terminal, (similar organisms as found before in sample No. 7).
11	Tapwater for Cholera C. Special: Maneckji Petit Mill (through Deputy Health Offi-cer E.).	6-8-12 at 11-45 a.m.	A few commas present microscopically but cultural tests negative for Cholera.
12	Tapwater Maneckji Petit Mill Tap No. 1 B. handed in by Dr. Sorab.	6-8-12 at 4-15 p.m.	No comma.
13	Tapwater Maneckji Petit Mill Tap No. 2 B. handed in by Dr. Sorab.		Comma present both microscopically and culturally (Indol present, etc.).
14	Tapwater Maneckji Petit Mill Tap No. 3 handed in by Dr. Sorab.		No comma.
15	Tapwater A. for Cholera obtained from Maneckji Petit Mill Tardeo through Deputy Health Officer E.	8-8-12 at 11-30 a.m.	Negative for Cholera.

Sd. S. L. JOSHI,
Municipal Analyst.

Statement showing the number of stools examined for cholera vibrio from January 1912 to 23rd September 1912.

Serial No.	Laboratory sample number.	Patient number.	Age.	Sex.	Caste.	Received from other than Hospital.	Dates of Attack.	Dates of Admission to Hospital.	Dates of Sample sent to Laboratory.	Reference to serial number if a specimen is repeated.	Remarks by A. R. Hospital of recovery or death.	Remarks by Municipal Analyst.
1	967	...	...	.....	.....	D. R. 1 ...	.....	.....	29th April 1912 ...	.....	...	Negative for cholera.
2	1118	802	35	Male ...	Christian ...	...	10th May 1912 ...	10th May 1912 ...	11th May 1912 ...	.....	Recovered ...	Both negative for cholera.
3	1119	801	30	Do. ...	Hindoo ...	...	6th May 1912 ...	10th May 1912 ...	11th May 1912 ...	.....	Died ...	
4	1139	...	...	.....	.....	D. R. 3 ...	.....	28th May 1912 ...	17th May 1912 ...	.....	...	Negative for cholera.
5	1179	899	20	Female ...	Hindoo ...	...	26th May 1912 ...	28th May 1912 ...	.....	.....	...	Negative for cholera.
6	1240	...	...	.....	.....	D. R. 3 ...	.....	.....	10th June 1912 ...	.....	...	Cholera like vibrio detected, cultural tests negative.
7	1344	981	45	Male ...	Hindoo ...	...	12th June 1912 ...	12th June 1912 ...	13th June 1912 ...	.....	Died 12th June 1912.	Negative for cholera.
8	1345	986	40	Do. ...	Musulman ...	...	12th June 1912 ...	13th June 1912 ...	13th June 1912 ...	.....	Recovered ...	Microscopically comma-shaped bacilli detected, cultural tests negative.
9	1356	999	30	Do. ...	Do. ...	...	13th June 1912 ...	14th June 1912 ...	14th June 1912 ...	.....	Died 19th June 1912.	Microscopically Vibrio and spirilli bacilli detected, cultural tests negative.
10	1407	1081	25	Do. ...	Hindoo ...	...	17th June 1912 ...	19th June 1912 ...	19th June 1912 ...	.....	Recovered ...	Positive for cholera.
11	1420	1090	35	Do. ...	Do. ...	...	19th June 1912 ...	20th June 1912 ...	21st June 1912 ...	.....	Do. ...	Both Negative for cholera.
12	1421	1091	25	Do. ...	Musulman ...	...	20th June 1912 ...	20th June 1912 ...				
13	1422	1092	30	Do. ...	Parsee ...	...	20th June 1912 ...	20th June 1912 ...	21st June 1912 ...	.....	Do. ...	Microscopically a few comma bacilli and vibrio seen, cultural tests negative.
14	1492	1153	30	Do. ...	Musulman ...	...	27th June 1912 ...	27th June 1912 ...	28th June 1912 ...	.....	Do. ...	Negative for cholera.
15	1493	1154	18	Female ...	European ...	...	27th June 1912 ...	27th June 1912 ...	28th June 1912 ...	.....	Do. ...	Few vibrio present; spirilli seen microscopically, cultural tests negative.
16	1686	1240	16	Do. ...	Hindoo ...	...	16th July 1912 ...	16th July 1912 ...	19th July 1912 ...	.....	Died ...	Negative for cholera.
17	1687	1243	18	Do. ...	Do. ...	...	12th July 1912 ...	17th July 1912 ...	19th July 1912 ...	.....	Recovered ...	Negative for cholera.
18	1688	1245	12	Do. ...	Do. ...	...	17th July 1912 ...	18th July 1912 ...	19th July 1912 ...	.....	Do. ...	A few spirilli seen microscopically; cultural tests negative.
19	1694	1207	28	Male ...	Christian ...	...	9th July 1912 ...	9th July 1912 ...	20th July 1912 ...	.....	Do. ...	Negative for cholera.
20	1695	1223	25	Do. ...	Hindoo ...	...	12th July 1912 ...	12th July 1912 ...	20th July 1912 ...	.....	Do. ...	A few comma bacilli seen microscopically, cultural tests negative.
21	1696	1238	25	Do. ...	Do. ...	...	15th July 1912 ...	16th July 1912 ...	20th July 1912 ...	.....	Do. ...	Both negative for cholera.
22	1697	1246	25	Do. ...	Do. ...	...	19th July 1912 ...	19th July 1912 ...	20th July 1912 ...	.....	Do. ...	
23	1735	1252	50	Do. ...	Do. ...	...	17th July 1912 ...	21st July 1912 ...	24th July 1912 ...	.....	Do. ...	Positive for cholera.
24	1736	1249	32	Do. ...	Do. ...	...	19th July 1912 ...	20th July 1912 ...	24th July 1912 ...	.....	Do. ...	Positive for cholera.
25	1737	1254	35	Female ...	Do. ...	...	21st July 1912 ...	22nd July 1912 ...	24th July 1912 ...	.....	Died ...	Microscopically comma-shaped baccilli and spirilli seen, cultural tests negative.
26	1738	1255	45	Male ...	Do. ...	...	21st July 1912 ...	22nd July 1912 ...	24th July 1912 ...	.....	Recovered ...	Negative for cholera.
27	1739	1259	9	Do. ...	Musulman ...	...	23rd July 1912 ...	24th July 1912 ...	24th July 1912 ...	.....	Died ...	Many spirilli and comma-shaped bacilli detected. Gave tests for cholera vibrio.

28	1804	1289	30	Male ...	Hindoo ...	...	1st August 1912 ...	2nd August 1912 ...	3rd August 1912 ...	43 12th August 1912.	Recovered ...	}	
29	1805	1290	30	Female ...	European ...	...	2nd August 1912 ...	2nd August 1912 ...	3rd August 1912 ...	.....	Do. ...	}	Cholera bacilli detected in all.
30	1806	1294	23	Male ...	Christian ...	...	31st July 1912 ...	2nd August 1912 ...	3rd August 1912 ...	.....	Do. ...	}	
31	1807	1280	22	Do. ...	Hindoo ...	...	30th July 1912 ...	31st July 1912 ...	3rd August 1912 ...	42 12th August 1912.	Do. ...		Negative for cholera.
32	1808	1288	35	Do. ...	Do. ...	...	27th July 1912 ...	1st August 1912 ...	3rd August 1912 ...	.....	Do. ...		Doubtful comma-like bacilli detected but negative for culture tests.
33	1809	1279	25	Do. ...	Musulman ...	...	30th July 1912 ...	31st July 1912 ...	3rd August 1912 ...	.....	Do. ...		Negative for cholera.
34	1873	1297	18	Do. ...	Hindoo ...	...	3rd August 1912 ...	3rd August 1912 ...	6th August 1912 ...	45 13th August 1912.	Do. ...		Microscopically comma and spirilli seen, could not get any pure culture.
35	1874	1284	30	Female ...	Musulman ...	...	1st August 1912 ...	1st August 1912 ...	6th August 1912 ...	.....	Do. ...		Negative for cholera.
36	1875	1315	23	Male ...	Christian ...	...	3rd August 1912 ...	5th August 1912 ...	6th August 1912 ...	.....	Do. ...		Numerous commas, vibrio and spirilli detected.
37	1909	1318	35	Do. ...	Hindoo ...	...	4th August 1912 ...	6th August 1912 ...	7th August 1912 ...	46 15th August 1912.	Do. ...	}	Comma bacilli detected in both.
38	1910	1319	12	Do. ...	Do. ...	...	4th August 1912 ...	6th August 1912 ...	7th August 1912 ...	.....	Do. ...	}	
39	1925	1304	40	Do. ...	Musulman ...	...	4th August 1912 ...	4th August 1912 ...	8th August 1912 ...	.....	Do. ...		A few comma-like bacilli seen microscopically; culture test negative.
40	1926	1307	22	Do. ...	Hindoo ...	...	4th August 1912 ...	4th August 1912 ...	8th August 1912 ...	.....	Do. ...		No commas.
41	1949	1313	35	Do. ...	Do. ...	...	5th August 1912 ...	5th August 1912 ...	10th August 1912...	.....	Do. ...		Negative for cholera.
42	1958	1280	2	Do. ...	Christian ...	...	1st August 1912 ...	1st August 1912 ...	12th August 1912...	.....	Died ...		A few comma-like bacilli present microscopically, negative for culture test.
43	1959	1289	30	Do. ...	Hindoo ...	...	1st August 1912 ...	2nd August 1912 ...	12th August 1912...	.....	Recovered ...		Isolated pure culture of commas. Indol present in 48 and 24 hours, Agglutination 1-100 present in 20 minutes.
44	1971	1284	...	...	...	...	...	...	13th August 1912...	.....	...	}	Both negative for cholera.
45	1972	1297	18	Male ...	Hindoo ...	...	3rd August 1912 ...	3rd August 1912 ...	13th August 1912 .	.....	Recovered ...	}	
46	1985	1315	...	...	...	...	...	...	15th August 1912...	Repeated 13th August 1912.	...		A few commas microscopically but cultural tests negative for cholera.
47	1986	1299	3	Female ...	Eurasian ...	...	2nd August 1912 ...	3rd August 1912 ...	15th August 1912...	Repeated 15th August 1912.	Recovered ...		Commas present, pure culture isolated; Indol present in 24 and 48 hours agglutination partial 1-100.
48	2020	1308	23	Male ...	Musulman ...	...	4th August 1912 ...	4th August 1912 ...	17th August 1912...	.....	Do. ...	}	Both negative for cholera.
49	2021	1318	35	Do. ...	Hindoo ...	...	4th August 1912 ...	6th August 1912 ...	17th August 1912...	.....	...	}	
50	2034	1330	30	Do. ...	Musulman ...	...	12th August 1912...	12th August 1912...	20th August 1912...	.....	Died ...	}	Do. do.
51	2035	1334	35	Do. ...	Hindoo ...	...	12th August 1912...	13th August 1912...	20th August 1912...	.....	Do. ...	}	
52	2074	1341	40	Do. ...	Do. ...	...	15th August 1912...	15th August 1912 ...	22nd August 1912 ...	.....	Recovered ...	}	A few comma-like bacilli seen microscopically with negative cultural tests for cholera for both.
53	2075	1332	27	Do. ...	Do. ...	...	12th August 1912...	12th August 1912 ...	22nd August 1912...	.....	Do. ...	}	
54	2080	1350	40	Do. ...	Parsee ...	...	20th August 1912...	22nd August 1912...	24th August 1912...	56 28th August 1912.	Do. ...		Smears showed comma bacilli, pure culture of commas isolated. Indol present in 24 and 48 hours. Agglutination 1-100 marked in 15 minutes.
55	2081	1351	22	Do. ...	Do. ...	...	20th August 1912...	22nd August 1912...	24th August 1912...	57	Recovered ...		Smears showed comma bacilli; pure culture of commas isolated. Agglutination 1 : 100 marked in 15 minutes. Indol positive in 24 and 48 hours.

Statement showing the number of stools examined for cholera vibrio from January 1912 to 23rd September 1912.

Serial No.	Laboratory sample register number.	Patient number.	Age.	Sex.	Caste.	Received from other than A. R. Hospital.	Date of Attack.	Date of Admission to Hospital.	Date of Sample sent to Laboratory.	Reference to serial number if a specimen is repeated.	Remarks by A. R. Hospital of recovery or death.	Remarks by Municipal Analyst.
56	2112	1350	40	Male ...	Parsee ...	...	20th August 1912...	22nd August 1912...	28th August 1912...	59 Repeated 28th August 1912.	Recovered ...	A few comma-like bacilli seen microscopically; cultural tests negative.
57	2113	1351	22	Do. ...	Do. ...	...	20th August 1912...	22nd August 1912...	28th August 1912...	60 Repeated 28th August 1912.	Recovered ...	Both negative for cholera.
58	2114	1357	21	Female ...	Hindoo ...	...	26th August 1912...	26th August 1912...	28th August 1912...	.....		
59	2128	1350	...	...	...	...	.....	.....	2nd September 1912	.....	...	Do. do.
60	2129	1351	...	...	...	...	.....	.....	2nd September 1912	.....	...	
61	2167	1370	45	Male ...	Parsee ...	...	5th September 1912	6th September 1912	7th September 1912	.....	Recovered ...	Very few comma-like bacilli seen microscopically but cultural tests negative.
62	2168	1371	35	Do. ...	Do. ...	...	5th September 1912	6th September 1912	7th September 1912	.....	Do. ...	Many comma-like bacilli present in pure culture. Indol test was faint. Agglutination 1 : 100 negative.

Summary.

Out of the 62 specimens of excreta received only 15 specimens were examined with positive results for cholera vibrio.

There were only 23 (included in 62) specimens from convalescent patients and out of these 23, only 2 specimens gave positive results for cholera vibrio.

NOTES ON VIBRIOS ISOLATED FROM VARIOUS SOURCES IN BOMBAY DURING THE RECENT OUTBREAK OF CHOLERA.

BY

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Introductory.—The chief interest which cholera-like vibrios possess centres in their possible relation to true-cholera vibrios. The relation may be considered both from the epidemiological and bacteriological side.

Cholera-like Vibrios in Water.—Lamb in collaboration with Haffkine examined the water of wells from 14 localities in Guzarat during the cholera epidemic of 1900. He found that comma-like bacilli were present in 7 out of 8 wells situated in localities where cholera was prevalent, whereas similar vibrios were found in only one out of six wells in localities free from cholera. Haffkine during the cholera season of 1894 found comma-shaped vibrios in 42 out of 46 tanks in Calcutta round which cholera existed at the time, but in only 11 out of 59 tanks round which the disease had not appeared during the preceding four months. Cayley made daily examinations of the waters of the Tansa, Vehar and Tulsi lakes during July, August and September 1900. During July, August, and the first-half of September when cholera was prevalent in the city, large numbers of vibrios were found in all three lakes, but they disappeared in the second half of September simultaneously with the disappearance of cholera from the city. It has been suggested that the presence of this morphological type in places where cholera exists and their relative scarcity in places free from the disease can be explained on the supposition that the whole class of vibrios, including true-cholera vibrios, require for their existence similar conditions.

Cholera-like Vibrios in Stools.—Up to the present no systematic study of non-agglutinating vibrios in the stools of persons in an infected country has been carried out. Certain isolated facts, however, point to their frequency during cholera epidemics. M'Laughlin during an epidemic in Manila in 1908 examined the stools of 376 contacts and isolated true cholera vibrios from 27 and non-agglutinating vibrios from 46.

Gotschlich during the pilgrimage of 1910-11 found 31 cholera and 23 non-cholera vibrios in the stools of 1,660 pilgrims arriving from the Hedjaz where cholera was raging. He emphasises the fact that all these vibrios were isolated from persons suffering from intestinal diseases.

Crendiropoulo examined the stools of 34,461 persons arriving in Alexandria by sea from infected places between the 17th August 1911 to the 31st January 1912. He found vibrios in the stools of 63 persons; of these 23 were agglutinating and 40 non-agglutinating.

Crendiropoulo made the important observation that during the height of an epidemic the majority of vibrios isolated were agglutinaters, but, in proportion as the epidemic declined, the carriers of true-cholera vibrios became more rare, while the non-agglutinaters increased. Finally, when the epidemic ceased no vibrios of either class were met with.

The only non-agglutinating vibrio isolated by Crendiropoulo during the month of August was from a woman who had close relations with a family in which two deaths from cholera occurred.

The above facts point to a certain epidemiological relationship between cholera-like vibrios and true cholera vibrios.

The whole question of the bacteriological diagnosis of cholera has recently been summed up by Crendiropoulo (Rapport sur l' examen des selles des voyageurs provenant des pays infectes de cholera, 1912) as follows:—None of the characters sometimes considered as specific can really serve as a criterion for the diagnosis of cholera vibrios. Virulence for the pigeon, liquefaction of gelatine, coagulation of milk, the presence of a single flagellum, the indol reaction, fixation of complement are either inconstant or common to some vibrios really saprophytic. Agglutination even, which at the moment appears to be the most useful diagnostic test is undergoing assaults which it resists with difficulty. The facts observed by Zirolia, and Zlagotoroff, and Crendiropoulo himself have shaken our confidence in the test. Zirolia has had in hand a vibrio isolated from the drinking water of a vessel, which was at first agglutinated by cholera serum, but gradually lost this faculty. Zlagotoroff has succeeded in transforming 45 of 82 non-agglutinating vibrios into agglutinaters by submitting them to various treatments. Even if agglutination indicates identity of race it teaches us nothing as to the cholera producing nature of the vibrio. The agglutinating vibrios which Ruffer and Gotschlich isolated at Tor during periods of freedom from cholera, and those which Mme. Pana yotatou has found in a patient suffering from diarrhœa remote from all possibility of infection and other facts just mentioned ought to make us circumspect. According to Crendiropoulo's personal opinion every carrier of vibrios whether agglutinating or non-agglutinating, who comes from an infected focus or may possibly have been infected from such a focus should be considered as a 'suspect'. On the other hand every carrier of vibrios agglutinating or not and not fulfilling the above conditions should not be considered as a source of danger.

The observations which form the subject of this paper are of some interest in connection with the questions just referred to.

Vibrios isolated from various sources in Bombay during the recent outbreak of Cholera.

During the recent outbreak of cholera in Bombay an examination of the stools of 32 and the intestinal contents of 2 patients diagnosed clinically to be suffering from cholera was carried out. In 26 cases the stools were those of patients within the first few days of their illness; the remaining eight stools were those of convalescents who had been attacked from 8 to 20 days previously. Vibrios were isolated from 13 of the 26 early cases. Of these 13 vibrios 11 agglutinated completely in dilutions of 1—1000 or over of high titre cholera serum. Of the remaining two one gave partial agglutination with cholera serum in dilutions of 1-20 and the other no agglutination. No agglutinating vibrio was isolated from the stools of the 8 convalescents, but from 2 non-agglutinating vibrios were obtained on the 8th and 10th day, respectively, of the patient's illness.

In many instances the stool was not received for examination until 12 or more hours after it had been passed which may account for the large number of negative results.

Details of the cases from which non-agglutinating vibrios were isolated.

Case 1280.—Arthur Road Hospital, adult male. Attacked on 30th July 1912, admitted on 31st July. Clinically this was a fairly severe case of cholera. No examination of the stool was made until 9th August. The appearance of stool was not noted. The peptone culture examined 18 hours after sowing showed almost a pure culture of vibrios. Sub-cultures on

Dieudonné slopes showed very numerous blue colonies of a cholera-like vibrio which did not agglutinate with cholera serum 1-50.

The serum of this patient on 14th August gave no trace of agglutination with his own vibrio (1280) even in a dilution of 1-10. On the other hand his serum agglutinated a recently isolated cholera organism (1255), completely in dilutions up to 1—150 and distinctly in a dilution of 1-300.

Case 1299.—A female child, age 4 or 5 years, from the Mazagaon Foundling Home. She was one of the last of a series of about 11 cases which occurred in the Home; of which 7 were fatal. She was attacked on 3rd August 1912 on which date she was sent to the Arthur Road Hospital. No examination of her stools was made in the beginning of the disease. Clinically her symptoms were those of a severe attack of cholera. The first stool was examined on 11th August. It consisted of yellowish mucous flakes in a clear fluid and microscopically showed numerous curved organisms, some like comma bacilli. The peptone culture showed numerous vibrios after 18 hours. Sub-cultures on Dieudonné slopes showed many blue colonies. These consisted of cholera-like vibrios which did not agglutinate with cholera serum in 1–50 dilution.

A second stool was examined on 14th August. It was a solid stool and contained much mucous. A non-agglutinating vibrio was again isolated.

A third stool was examined on 20th August. This was a solid normal stool. The examination for vibrios gave a negative result.

Agglutination.—Patient's serum on 14th August did not agglutinate the organism (1299) isolated from her own stool. The effect of her serum on a cholera organism was undetermined owing to the strain used (L. I. P. M.) being auto-agglutinating.

Case, Jehangir.—An adult Parsee inmate of the Colaba Lunatic Asylum. Attacked on 5th September 1912 with vomiting and diarrhœa, suppression of urine, cold extremities, and collapse. Stool examined on 6th September. A fluid stool yellowish in colour containing mucous flakes. Microscopically, many curved bacilli, some like cholera vibrios were found. The peptone culture after five hours showed many vibrios. Subcultures on Dieudonné medium developed numerous blue colonies of a cholera-like vibrio not agglutinating with 1-50 cholera serum.

Agglutination.—Patient's serum on 12th September agglutinated his own vibrio completely in a dilution of 1-10, but not in any higher dilution. Normal human serum gave no agglutination with the same organism (Jehangir) in dilutions of 1-10 or over. The patient's serum agglutinated a cholera vibrio (Nusserwanji I) completely in a dilution of 1-50 and partially in a dilution of 1-150. On 9th October patient's serum still agglutinated his own vibrio in a dilution of 1-10 but its agglutinative power for cholera had diminished as when tested against a cholera vibrio (1091), it gave only slight agglutination in a dilution of 1-25 and none in 1-50 or higher dilutions.

Case, Jijibhoy.—An adult Parsee inmate of the Colaba Lunatic Asylum attacked with vomiting and diarrhœa on 5th September 1912. His symptoms were mild, no suppression of urine and no collapse. He was convalescent on

8th September. His stool was examined on 7th September. It was a fluid feculent stool. A non-agglutinating vibrio was isolated by cultivation in peptone medium and subculturing on to Dieudonnè slopes.

On 12th September, the patient's serum agglutinated his own vibrio (Jijibhoy), partially in dilutions of 1-10 and 1-20, but not at all in 1-50. The same serum diluted to 1-20 produced only a trace of agglutination with a cholera vibrio (Nusserwanji I).

These two cases, Jehangir and Jijibhoy, were the last of a series of 7 cases clinically resembling cholera which occurred among the Parsee inmates of the Colaba Lunatic Asylum between 18th August and 5th September. Three of these cases died. Agglutinating vibrios were isolated from the stools or intestinal contents of four. Material from the remaining case was not examined bacteriologically as death occurred within a few hours of attack.

A vibrio isolated from an old stool.—In addition to the 15 vibrios isolated from recently passed stools, a non-agglutinating vibrio (Nusserwanji II) was obtained from an old cholera stool which had been kept for 16 days in a sterile petri dish in the Laboratory. An agglutinating vibrio (Nusserwanji *I*) had been isolated from the same stool when fresh.

Non-agglutinating vibrios were also isolated from the following other sources :—

Wells	2
Tank	1
Sewage	1
Cockroach	1

Both the vibrios isolated from wells were kindly sent by Dr. Turner, Health Officer, Bombay Municipality. One (No. 1406) was obtained from a well in a mosque which was in the centre of the then infected area, the other (No. 1507) from a well in Mazagaon which was not at the time infected. A case of cholera had, however, occurred some months previously in the compound in which the well is situated. The tank from which a vibrio was isolated is situated close to the Arthur Road Hospital in which cholera cases were being treated and is exposed to fæcal contamination from several latrines around it. At the time the vibrio was isolated several cases of cholera occurred in a Municipal Chawl in the vicinity of the tank.

The sewage furnishing a vibrio was obtained from a trap in connection with a drain from the water closets in the Colaba Lunatic Asylum where cases of cholera were occurring at the time.

The cockroaches from which a non-agglutinating vibrio was isolated were obtained in a man-hole in connection with the same drain in the Colaba Lunatic Asylum. The vibrio was obtained from the peptone water in which the cockroaches had been washed and is probably the same as that found in the sewage. We did not succeed in obtaining a vibrio from the contents of the gut of any of the same (6) cockroaches.

BACTERIOLOGICAL.

The method of examination.—In most of the stools examined curved bacilli varying in size and form were seen, but usually they were neither numerous nor typical enough to enable a provisional diagnosis of cholera to be made. Slender spirilla with two or more curves which stained faintly with carbol fuchsin were of common occurrence. Sometimes these occurred in enormous numbers and in felted masses, at other times they were few and occurred singly or in groups of 2 or 3. These spirilla were most frequently seen in typical cholera stools from which cholera vibrios were subsequently isolated.

Cultural methods.—A preliminary cultivation in peptone water was always made. About 1 c. c. of the stool or intestinal contents was sown in 50 c. c. of special peptone medium containing :—

Peptone	... ...	10·0
Salt	... ...	10·0
Potassium nitrate	...	0·1
Sodium carbonate	...	0·2
Distilled water	...	1,000

When possible the peptone culture was examined 4–6 hours after sowing. If vibrios were seen microscopically subcultures were made on to slopes of Dieudonné medium. When the peptone water showed no vibrios after 4–6 hours a subculture was made with a tube of peptone water and both this and the original culture were examined next day. When no vibrios were seen in the peptone after 18 to 20 hours the result was considered negative. In examining water samples for vibrios 10 c. c. of a ten per cent. peptone solution was added to 100 c. c. of the water and the subsequent procedure was the same as for stools. That Dieudonné's medium possesses advantages over ordinary agar for the isolation of vibrios was shown by some tests in which subcultures were made from the same peptone growth on to both of these media ; in each test the number of colonies developing on the Dieudonné medium was greater than on the agar.

It was not possible during the short time I was employed on the cholera enquiry to make a complete study of the characters of all the vibrios isolated. A summary of these characters, in so far as they were determined, is given in Table I, in which are also included for purposes of comparison four laboratory strains of cholera.

Morphology.—The form of all the vibrios was not the same, but the difference in this respect between the agglutinating and non-agglutinating was no greater than between individual vibrios of the same group or even between different cultures of the same vibrio. The most common form in both groups was a short fairly thick slightly curved vibrio. All were motile and did not stain with Gram. Of five agglutinaters and 6 non-agglutinaters stained for flagella all were monociliate.

TABLE I.

Serial No.	Name of Vibrio.	Source of Vibrios.	Indol.	Agglutination.	Pfeiffer's test.	Fixation test.	Hæmolysis.*		
							2 hours.	24 hours.	48 hours.
		AGGLUTINATING VIBRIOS.							
1	1091	Cholera stool	+	2,000	+	+	—	—	—
2	1092	Do.	+	4,000	Indefinite.	..	—	—	—
3	1407	Do.	+	1,000	..	..	..	..	..
4	1190	Do.	+	2,000	+	+	—	—	—
5	1255	Do.	+	1,000	..	..	—	—	—
6	1259	Do.	..	1,000	..	..	—	—	—
7	No. 3	Do.	..	1,000	..	..	—	—	tr.
8	DeSouza	Do.	..	1,000	..	..	—	—	tr.
9	Billimoria	Do.	.	1,000	..	..	—	—	tr
10	Nusserwanji I	Do.	+	1,000	..	..	—	—	tr.
11	Delaney	Do.	..	1,000	..	..	—	—	tr.
12	Dosabhoy	Contents of ileum of a cholera case	+	1,000	..	+	—	—	tr.
13	Bombay cholera	Laboratory strain of cholera...	..	1,000	..	..	—	—	—
14	St. George's	Do. do. ...	..	1,000	..	..	—	—	—
15	L. I. P. M.	Do. do. ...	+	1,000	Indefinite.	..	—	—	—
16	Yerroda	Do. do. ...	..	1,000	..	..	—	—	—
17	M.	Contents of ileum of a cholera case	+	1,000	Indefinite.	..	—	++	..
		NON-AGGLUTINATING VIBRIOS.							
18	1406	Well	+	50 sl	—	..	—	+	+
19	1507	Do.	+	20	—	—	++	++	++
20	1289	Stool of a convalescent cholera case	+	50 sl.	—	..	+	++	++
21	1299	Do. do. ...	+	50 sl.	—	..	+	++	++
22	DeLisle Road	Tank	+	100 sl.	—	..	++	++	++
23	Jehangir	Stool of a case with symptoms of cholera	+	—	—	—	++	++	++
24	Jijibhoy	Do. do. ...	+	—	—	..	..	.	..
25	Colaba Sewage	Sewage	+	100 sl.	—	..	..	..	..
26	Cockroach	Cockroach found in sewage ...	+	—	..	..	..	..	..
27	Nusserwanji II	Old stool of a cholera case, Nusserwanji, which had been kept for 16 days ...	+	—	—	—	—	+	+

* Of goat's red blood cells.
sl. = Slight agglutination.
\+ = Partial hæmolysis.
++ = Complete „
tr. = trace of „

Peptone water.—The growth in peptone water was not characteristic. All the vibrios produced uniform turbidity. Several of the non-agglutinaters produced a definite scum on the surface of the peptone medium in 24 hours, while none of the agglutinaters did so before 48 hours.

Agar.—On agar the colonies of the agglutinaters could not be differentiated from those of non-agglutinaters. Both were bluish, opalescent, and fairly translucent in young cultures.

Indol reaction.—This test was applied to 9 of the agglutinaters and all the non-agglutinaters. The result was always positive after 24 hours' growth in peptone water. The colour which varied from a slight pink to a marked red developed on the addition of sulphuric acid alone. On the whole the reaction was more strongly marked among the non-agglutinaters.

Fermentation of sugars.—The changes produced by 11 agglutinaters and 9 non-agglutinaters in a 1 per cent. peptone medium coloured with litmus and containing 1 per cent. of the sugar were studied. All produced fermentation of glucose, lævulose, maltose, galactose, saccharose, mannite, dextrin and starch with the formation of acid but no gas in 24 hours at 37°C and did not ferment arabinose, raffinose, sorbit, dulcit, inulin, and adonit. None of the vibrios produced acid in lactose within the first 3 days. Two agglutinaters (L.I.P.M. and 1091), however, fermented this sugar on the 4th and 9th day, respectively, as did one non-agglutinater (1280) on the 9th day. Some vibrios both of the agglutinating and non-agglutinating groups produced bleaching without any acid reaction in the medium containing lactose. This change took place from the 3rd day onwards. Many of the non-agglutinaters differed from the agglutinaters in producing a secondary formation of alkali in lævulose after the early acid fermentation. This change occurred from the 3rd to the 7th day. One non-agglutinating strain (Jehangir) after preliminary acid fermentation produced alkali in dextrin on the 7th day.

Virulence for pigeons.—The test was carried out with one agglutinater (1091) and two non-agglutinaters (1406 and 1507). The dose was 1 loop of a 20-hour agar culture suspended in 1 c. c. of saline and it was injected deeply into the pectoral muscle. The pigeons inoculated with cultures 1091 and 1406 died within 24 hours and 3rd pigeon within 48 hours. Cultures of vibrios were obtained from the heart-blood of all three pigeons.

Agglutination.—The test was carried out macroscopically. The method employed was the usual one with Wright's pipettes. The agar cultures were 20-24 hours old. The results were noted next day after the tubes had stood at room temperature. The serum used was a highly agglutinating serum obtained from the Swiss Serum and Vaccine Institute, Berne. Its nominal titre was 1-10000. The results of the agglutination tests are shown in Table I. Many of the agglutinaters were not tested in serum dilutions between 1-1000 and 1-5000. Some of the vibrios included in the group of non-agglutinaters gave feeble agglutination in serum dilutions up to 1-100, but none in any higher dilutions.

None of the members of the cholera-like group acquired any appreciable increase in agglutinability after repeated sub-culturing on agar extending in some cases over 3 months. Two vibrios, 1406 and 1507, were passed once through a guinea-pig and once through a pigeon without their agglutinability by cholera serum undergoing any change. One old laboratory strain of cholera, (L. I. P. M.) gave anomalous results with the specific agglutinating serum as

will be seen in the subjoined table. Occasionally vibrios, which agglutinated completely in serum dilution of 1-500 and 1-1000, failed to agglutinate in the lower dilutions, 1-50 and 1-100.

Strain L. I. P. M. Cholera. Date.	Serum Dilutions. 1-20	1-50	1-100	1-200	1-500	1-1000	1-5000	Salt Control.
22nd June	.....	.....	—	—	—	—	.....	—
28th „	.....	—	—	—	—	—	.....	—
2nd July	+	++	++	++	++	.....	.....	tr.
17th August	.....	++	++	++	++	++	++	—*
3rd October	.....	sl.	+	.....	+	—	—	—

* This control showed no agglutination when the reading was taken after 20 hours, but 2 hours later it had completely agglutinated.

++ = Complete agglutination. + = Partial agglutination. — = No agglutination.

sl. = Slight tr. Trace.

Hœmolytic power.—The question whether true-cholera vibrios produce hæmolysis has been the subject of considerable discussion. Ruffer in 1906 examined a series of agglutinating strains and found that, with the exception of the El Tor vibrios none were hæmolytic. The majority of the cholera vibrios he tested were old strains isolated during a cholera epidemic in El Tor in 1902. Kolle and others, also using old cultures, demonstrated that certain cholera strains possess hæmolytic properties. Kraus working with fresh cholera cultures failed to find any that hæmolysed. Mc'Laughlin and Whitmore also failed to find any hæmolytic strains among the agglutinating vibrios isolated in the Phillipines. Crendiropoulo found that 2 out of 19 agglutinating vibrios, which he isolated in Alexandria in 1911, hæmolysed.

The hæmolytic action of our vibrios was tested by the method devised by Crendiropoulo. A small quantity of an 18-24 hours agar culture was rubbed up in 0·5 c. c. of normal saline and to this emulsion 0·5 c. c. of a 5 per cent. suspension of well-washed blood cells was added. The tubes were then placed in the incubator and the results noted after 2, 24 and 48 hours.

Two series of tests were carried out—one with goat's and the other with human red blood cells. The results are shown in Table III. None of the 17 agglutinaters which were tested hæmolysed goat's red blood cells in 24 hours at 36° C. Six produced only a trace and one definite, but still incomplete hæmolysis after 48 hours. The last was a quite recently isolated strain.

The results with human red blood cells were very different. Twelve agglutinating vibrios were tested, out of which 11 produced complete or partial hæmolysis in 24 hours. The strain which did not hæmolyse was an old laboratory one. The hæmolytic power of 7 non-agglutinaters was tested against goat's corpuscles and three of the same strains were also put up with human blood cells. Hæmolysis of both kinds of cells was produced in 24 hours. Only one strain, No. 1406, gave divergent results in two successive experiments (*vide* Table No. III).

It would appear from these results that cholera vibrios produce hæmolysins, which are to some extent specific for human blood cells.

Table III.

Hæmolytic Power.

Name of Culture.	Interval of time since the Vibrio was isolated.	HÆMOLYSIS.				
		Goat's red blood cells.			Human red blood cells.	
		2 hours.	24 hours.	48 hours.	24 hours.	48 hours.
AGGLUTINATERS.						
1091	3 months.	—	—	—	++	++
1092	Do.	—	—	—	+	++
1190	Do.	—	—	—	+	++
1255	2 months.	—	—	—	++	++
1259	Do.	—	—	—	+	++
No. 3 stool	Do.	—	—	tr.	++	++
De Souza...	7 weeks.	—	—	tr.	..	..
Delaney	5 Do.	—	—	tr.	..	..
Nusserwanji I	Do.	—	—	tr.	+	++
Billimoria	Do.	—	—	tr.	..	..
Dossabhoy	4 weeks.	—	—	tr.	++	++
L. I. P. M.	Old laboratory strain.	—	—	—	tr.	sl.
Yerrowda...	Do.	—	—	—	..	..
Bombay	Do.	—	—	—	+	++
St. George's	Do.	—	—	—	..	..
Baroda	Do.	—	—	—	++	++
M.	3 days.	..	—	+	++	++
NON-AGGLUTINATERS.						
*1406	3½ months.	—	+	+	++	..
1507	3 Do.	++	++	++	++	..
DeLisle Road	2 Do.	++	++	++	++	..
1280	2 Do.	+	++	++	..	..
1299	2 Do.	+	++	++	..	..
Jehangir	1 month.	+	++	++	..	..
Nusserwanji II	1 Do.	—	+	+	..	..

* In a second experiment 1 week later, this vibrio gave only slight hæmolysis with goat's red blood cells after 48 hours.

++ = Complete hæmolysis. sl. = Slight hæmolysis.

+ = Partial „ tr. = A trace of „

. . = No experiment made.

Pfeiffer's reaction:—Guinea-pigs were used for this test. The serum was the Berne highly agglutinating serum. In one test the serum of a recovered cholera patient was used as a control.

Table No. II. gives the details of the tests. Out of five tests with agglutinating vibrios, in two a definitely positive reaction was obtained, in the remaining three the control guinea-pigs which received culture without serum survived.

None of the nine non-agglutinaters reacted positively to the test.

With the exception of one strain, Nusserwanji II, all the latter were highly pathogenic to guinea-pigs, more so than were true cholera vibrios. Thus only 1 out of 5 of the latter was fatal to a guinea-pig in a dose of $\frac{1}{4}$ loop, whereas 7 out of 9 of the non-agglutinaters killed a guinea-pig in the same dose.

TABLE II.

Result of Pfeiffer's Reaction.

Name of Vibrio.	Specific Serum.		Serum normal, Horse ·001, Culture 1 loop-ful.	Serum of cholera patient 1091, ·003 culture 1 loop.	No serum culture ½ loop.
	·001 Culture 1 loop.	·002 Culture 1 loop.			
1091	Complete bacteriolysis. Guinea-pig survived.	Complete bacteriolysis. Guinea-pig survived.	.....	.....	No bacteriolysis. Guinea-pig died within 24 hours.
L. I. P. M. ...	Guinea-pig survived.	Partial bacteriolysis. Guinea-pig survived.	.....	.....	No bacteriolysis. Guinea-pig survived.
1406	No bacteriolysis. Guinea-pig died within 24 hours.	No bacteriolysis. Guinea-pig died within 24 hours.	.....	.....	No bacteriolysis. Guinea-pig died within 3 days.
1092	.....	Incomplete bacteriolysis. Guinea-pig died.	.....	Incomplete bacteriolysis. Guinea-pig survived.	No bacteriolysis. Guinea-pig survived.
1507	.....	No bacteriolysis. Guinea-pig died.	.....	No bacteriolysis. Guinea-pig died.	No bacteriolysis. Guinea-pig died.
1190	Almost complete bacteriolysis. Guinea-pig survived.	Complete bacteriolysis. Guinea-pig survived.	No bacteriolysis. Guinea-pig died after 24 hours.	.....	No bacteriolysis. Guinea-pig survived.
M. ...	Almost complete bacteriolysis. Guinea-pig survived.	Almost complete bacteriolysis. Guinea-pig survived.	.....	.....	No bacteriolysis. Guinea-pig survived.
DeLisle Road...	No bacteriolysis. Guinea-pig died within 24 hours.	No bacteriolysis. Guinea-pig died within 24 hours.	.....	.....	No bacteriolysis. Guinea-pig died within 24 hours.
1280	Do. ...	Do. ...	.....	.....	Do.
1299	Do. ...	Peritonial fluid not examined. Guinea-pig died within 24 hours.	.....	.....	Partial bacteriolysis. Guinea-pig died within 48 hours.
Nusserwanji II.	No bacteriolysis. Guinea-pig survived.	No bacteriolysis. Guinea-pig survived.	.....	.....	Complete bacteriolysis. Guinea-pig survived.
Jijibhoy ...	No bacteriolysis. Guinea-pig died within 24 hours.	No bacteriolysis. Guinea-pig died within 24 hours.	.....	.....	Complete bacteriolysis. Guinea-pig died within 24 hours.
Jehangir ...	Do. ...	Do. ...	.....	.....	No bacteriolysis. Guinea-pig died within 24 hours.
Colaba Sewage.	Do. ...	Do. ...	.....	.....	Do.

Complement fixation.—Considerable differences of opinion exist as to the value of the complement fixation in differentiating true cholera from cholera-like vibrios.

Ruffer and Crendiropoulo, who studied 42 strains of vibrios in Egypt in 1905, found considerable irregularities in their behaviour to the test. Certain vibrios (El Tor) reacted positively to the agglutination and Pfeiffer's tests, but failed to fix complement in the presence of cholera immune serum, while, on the other hand, several strains which did not agglutinate or give a positive Pfeiffer's reaction gave a positive result with the fixation test. In a later study of 52 vibrios isolated from the passengers and crews of vessels arriving in Alexandria in 1911 from infected ports, Crendiropoulo obtained positive results only with agglutinaters. Other observers, notably Haendel, found that immune cholera serum fixed complement equally well with pseudo-cholera and true-cholera vibrios.

Method.—In preparing the extracts of the vibrios we followed the method described by G. Dean for the preparation of extracts of organisms of the paratyphoid group. Roux bottles of nutrient agar were freely sown and incubated for 4-5 days. The growth of each bottle was then emulsified in 20 c.c. of distilled water and transferred to test tubes. The emulsion was first heated to 60°C. for an hour and was then frozen hard, allowed to stand at room temperature and shaken for 6-8 hours. The freezing and shaking was repeated on two successive days. The emulsion was now centrifuged and the clear supernatant fluid pipetted off and used as the antigen. After several preliminary experiments to determine what dilutions of extract and serum were most suitable the following scheme was adopted :—

Scheme of Complement Fixation Experiments.

Tube No.	Extract (1-20).	Cholera serum (1-80).	Normal horse serum (1-80).	Guinea-pig's serum 30 %.	Hæmolytic serum (1-20).	Normal saline.		Human red blood cells (10 % suspension).
1	0·5 c. c.	0·5	.....	0·1	0·1	.....	1 hour's incubation at 36° C.	0·1
2	0·5 c. c.	0·5	.....	0·1	.....	0·1		0·1
3	0·5 c. c.	.....	0·5	0·1	0·1	.....		0·1
4	0·5 c. c.	.....	.....	0·1	0·1	0·5		0·1
5	.....	0·5	.....	0·1	0·1	0·5		0·1
6	.....	.....	.....	0·1	0·1	1·0		0·1

The hæmolytic serum was that of a rabbit immunised against human red blood cells.

Tube No. 2 in the above scheme is a control tube containing no hæmolytic serum and is necessary when using extracts of vibrios producing simple hæmolysis which do not require complement for their action.

Three non-agglutinating and three agglutinating vibrios were tested by the above method. All the agglutinaters completely fixed complement in the presence of specific cholera serum, while none of the non-agglutinaters gave even partial fixation.

Summary.

1. Non-agglutinating vibrios alone were isolated from the stools of 4 patients with symptoms of cholera. Two of these vibrios were agglutinated by the patient's serum in low dilutions, 1-10 and 1-20, while two were not agglutinated at all by the corresponding serums. The serums of two of the four

patients agglutinated cholera bacilli in dilutions of 1-150 or upwards, that of one did not agglutinate at all and the fourth patient's serum gave an indefinite result.

2. Non-agglutinating vibrios were also isolated from an old cholera stool, two wells, a tank and a sample of sewage.

3. No constant differences were observed between the morphological or cultural characters of the agglutinaters and the non-agglutinaters.

4. All the non-agglutinaters except one were pathogenic to guinea-pigs when given intra-peritoneally. Two non-agglutinaters and one agglutinater which were tested as regards their virulence for the pigeon gave positive results.

5. All the agglutinaters failed to hæmolyse goat's red blood cells in 24 hours at 36° C, while all the non-agglutinaters which were tested produced complete or marked hæmolysis. On the other hand under the same conditions of experiment both the non-agglutinaters and agglutinaters which were tested hæmolysed human red blood cells.

6. Three agglutinating vibrios tested for complement fixation gave a positive result, while with three non-agglutinaters the result of the test was negative.

Conclusions.—During a recent outbreak of cholera in Bombay we have isolated cholera-like vibrios from the stools of "cholera" patients, from the water of two wells and a tank exposed to fæcal contamination and from sewage. These vibrios are indistinguishable from true cholera vibrios morphologically and by their cultural characters, but do not react with a specific cholera serum. Further, they differ from true cholera vibrios in some of their hæmolytic properties. The identity or otherwise of the various non-agglutinating vibrios has not yet been determined by reciprocal serum tests, but rabbits are being immunised with this object. The present observations supply no evidence as to whether cholera-like vibrios exist in persons, water or sewage not exposed to cholera infection.

Even if we accept the observations of Zlagotoroff and others that vibrios may under certain conditions lose or acquire the power of agglutinating with cholera serum, the difference in hæmolytic properties observed between the non-agglutinating and the true cholera vibrios isolated in Bombay makes it difficult to believe that the former are cholera vibrios which have become modified by their environment.

Note on the vitality of cholera vibrio in unsterilised water.

Two experiments have been carried out :—

Experiment I.—On 6th July 1912, four drops of a cholera stool (from which an agglutinating vibrio was isolated), were added to tap water contained in a sterile Winchester quart bottle. The stool had been passed about 24 hours previously. The bottle was kept screened from light. Five hours after the stool had been added 90 c. c. of the water was removed with a pipette without disturbing the sediment and was mixed with 10 c. c. of 10 per cent. peptone medium. After 20 hours films prepared from the peptone growth showed many typical vibrios. By subculture on Dieudonne's medium several colonies of an agglutinating vibrio were obtained. An agglutinating vibrio was recovered by a similar procedure from samples of 100 c. c. taken 27 and 53 hours after the stool was added. From a 4th sample taken four days after contamination no vibrios were recovered. On the 11th day a fifth sample also gave negative results.

The vibrios isolated from the water on the 1st, 2nd and 3rd day were found to have undergone no change in their agglutinability with cholera serum.

Experiment II.—To a Winchester quart nearly full of water obtained from a disused well in the Parel compound 15 drops of a cholera stool (1259), containing agglutinating vibrios were added. We failed to isolate any vibrio from a sample of 100 c. c. of the water 48 hours after the stool had been added. Ten days later three samples were put up in peptone medium of different degrees alkalinity but no vibrios developed in any of the cultures.

Agglutinating power of Cholera Patient's Serum.

The agglutinating power of the serums of several convalescents from cholera was tested. The results which are given in tabular form below point to a marked loss in agglutinating power about a month after convalescence.

Agglutination of Cholera vibrios by Patient's Serum.

Serum.	Interval in days between dates of attack and agglutination test.	Culture.	Agglutination.	
978	16	1091	+ 1-200	— 1-500
1091	11	1091	tr. 1-200	— 1-500
1092	11	1091	+ 1-500	— 1-1000
1280	15	1255	+ 1-300	— 1-600
Nihal	14	Nusserwanji I.	+ 1-200	tr. 1-300
Ota	8	do.	+ + 1-300	..
Mah' Tilam	13	do.	+ + 1-300	sl. 1-450
Ali Umar...	13	do.	+ + 1-300	..
Jehangir	8	do.	+ 1-150	— 1-300
„	34	1091	+ + 1-10	tr. 1-25
Billimoria	49	1091	+ 1-100	— 1-200
Nusserwanji	51	Dossabhoy.	+ 1-25	tr. 1-50

+ + = Complete agglutination.
+ = Partial „
sl. = Slight „
tr. = Trace of „
.. = No experiment done

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PART VII.

FEVERS AND INFECTIOUS DISEASES.

ALL-INDIA SANITARY CONFERENCE—MADRAS—NOVEMBER 1912.

DYSENTERY,

PROBLEMS AND PROPOSALS.

BY

Captain J. Cunningham, I.M.S., and Major W. F. Harvey, I.M.S.

Definition.—Various definitions are possible according as we take locality, manifestations, cause, etc., as our basis; so we take as the most convenient as well as the most practical, the combined definition by clinical manifestations and locality.

Dysentery is a disease of the lower bowel which manifests itself by frequent dejections and straining, with blood and mucus in the stools. Such a definition affords a ready and sufficient means of the diagnosis of clinical dysentery.

Causation.—It is when we are called to decide upon the cause of the syndrome of dysentery that our difficulties begin. The problem arises as to the manifoldness or the oneness of the cause, and as to the differentiation of the causes. There can be, we think, little doubt as to manifold causation. Dysentery has been recognised as a manifestation of certain diseases—malignant disease, tuberculosis, kala azar, helminthic infection and probably others. Within comparatively recent years, however, two causes have been differentiated which may, in the sense that they have their significance only in the dysentery which they produce, be called specific causes. They give rise to the forms known as amœbic dysentery and bacillary dysentery. It is very largely in connection with these that our problems arise. Their differentiation from one another and from the other conditions which can give rise to dysentery is most important for both the treatment and the prophylaxis of this distressing disease.

The differentiation might seem to be easy—the dysentery showing the presence of dysentery bacilli is bacillary, that showing the presence of specific amœbæ is amœbic. But all is not such plain sailing as would be implied by this statement. We want to be instructed as to what is the amœba of dysentery, what are the bacilli of dysentery, whether both amœbæ and bacilli may combine to give rise to the disease and whether the one condition may or may not pass into the other. We should like to know, in connection with the delimitation of cause, what is the relation of chronicity to particular findings. It is, for example, said to be difficult to find the bacilli of bacillary dysentery in chronic cases. We must then in the amœbic case—said to be characteristically chronic—not lay too much stress on the absence of bacilli as these would be likely to be absent in any case.

We should also make certain that the bacillary form, become chronic, does not then begin to show the presence of amœbæ.

In this matter of the determination of casuation of a disease in which it may not be possible always to isolate a causal agent it becomes necessary to fall back upon indirect methods of determination. These may consist in (1) tests applied with the help of known and isolated causal organisms, (2) tests dependent on the associations, other than specific cause, with one or other form of the disease such as the characteristic chronicity, degree of leucocytic exudation and form of successful treatment.

Take the first of these in more detail.

Tests applied with the help of known and isolated causal organisms.—We admit, say, the existence of a bacillary dysentery and, therefore, of a bacillus of dysentery. Everyone is acquainted with the great use which is made of serum reactions not only for the diagnosis but for the differentiation of the Typhoid fevers.

We should expect, then, that a serum reaction, such as that of agglutination, would be of service in the delimitation of bacillary dysentery from the other dysenteries and also as a means for the further sub-division of this form of the disease.

In any group of dysenteries taken at random, simply on the basis of the description of the syndrome, we should expect that a certain number would be or would have been of the bacillary type and that such a test as that described would be evident in proportion to the prevalence of that type. The expectation would be capable of verification most satisfactorily in cases of actual dysentery.

This has been done by various observers and the results are variously given. We ourselves have not had dysentery cases to examine but have taken a random sample of a certain population and proceeded to examine the serum reactions of this sample to a variety of known dysentery organisms. The results were recorded and afterwards compared with the history or absence of history of dysentery afforded by the individuals with an idea that an agglutination reaction might be demonstrable in those with a recent history of bacillary dysentery. Incidentally a still larger number of individuals of the same population were interrogated as to the occurrence of dysentery among them and thus some idea, within the limits of credibility to be placed on the answers, was obtained as to the prevalence of the disease. The population was, if not an actually random sample of the population of India, at least a very varied sample of that of Northern India.

The agglutination tests are shown in the following tables in conjunction with the presence or absence of a history of dysentery.

The figures shown in Table I were obtained from the case sheets of the patients who attended the Pasteur Institute of India during the months of April, May, June and July 1912.

TABLE I.

Showing the prevalence of dysentery in an *unselected* population.

The table is divided into two subdivisions, the first (A) showing the population divided up according as they give a positive or negative history of dysentery. Those who give a positive history are also divided according to the period of time which has elapsed since their last attack.

Subdivision B. shows the age distribution of the population.

A.		B.			
Dysentery Incidence.		Age distribution.			
Date of last attack in months up to 1 year & in years up to 20 years.	No. of cases.	Age in years.	No.	Age in years.	No.
1 month ago	35	1	3	36	3
2 months ago	24	2	4	37	7
3 „ „	13	3	9	38	11
4 „ „	6	4	9	39	0
5 „ „	5	5	17	40	50
6 „ „	26	6	21	41	1
7 „ „	4	7	25	42	9
8 „ „	1	8	33	43	1
9 „ „	2	9	24	44	5
10 „ „	1	10	26	45	24
11 „ „	0	11	21	46	2
1 year ago	39	12	25	47	1
2 years ago	42	13	12	48	3
3 „ „	25	14	19	49	2
4 „ „	9	15	18	50	37
5 „ „	24	16	15	51	0
Carried over ...					

Dysentery Incidence.		Age distribution.			
Date of last attack in months up to 1 year & in years up to 20 years.	No. of cases.	Age in years.	No.	Age in years.	No.
Brought forward ...					
6 years ago	7	17	6	52	2
7 „ „	8	18	33	53	0
8 „ „	2	19	12	54	1
9 „ „	0	20	37	55	7
10 „ „	16	21	13	56	2
11 „ „	1	22	45	57	2
12 „ „	4	23	15	58	1
13 „ „	0	24	17	59	0
14 „ „	2	25	56	60	14
15 „ „	8	26	20	64	1
16 „ „	0	27	11	65	2
17 „ „	0	28	33	66	1
18 „ „	0	29	3	68	1
19 „ „	0	30	60	70	2
20 „ „	3	31	4	75	1
		32	16		
Over 20 „ „	4	33	4		
		34	5		
Never	592	35	39		
Total ...	903	...	...	...	903

TABLE II.

Showing the agglutinating power of the serum taken from an *Unselected* population when tested with a series of strains of B. Dysenteriæ.

Organisms.	Serum Dilutions with number of cases under each.													Total No. of cases.
	−5	+5	10	15	20	25	30	40	80	100	120	140	160.	
B. Shiga 1 ...	45	61	25	10	10	2	2	1	1	0	0	0	0	157
„ 2 ...	40	43	24	4	3	1	2	1	0	0	0	0	0	118
„ 3 ...	36	38	28	5	3	3	3	1	0	0	0	0	0	117
B. Flexner 1	37	33	38	29	20	3	55	44	1	0	0	0	0	170
„ 2	2	32	43	36	20	15	15	4	2	1	0	0	0	170
„ 3	5	31	40	35	23	10	17	6	2	0	0	0	0	169
B. Strong ...	5	4	4	14	12	12	34	20	11	0	1	0	0	119
B. Y. His ...	21	30	24	16	17	9	2	0	0	0	0	0	0	119

Explanation of Table II.

The reactions to three strains of Shiga (Shiga 1, 2, 3,), three strains of Flexner (Flexner 1, 2, 3,) and to a single strain each of B. Strong and B. Y. His are shown. Of these cultures Shiga 1 and 2 and Flexner 1 are strains of known age, Shiga 1 having been isolated in April 1912, Shiga 2 in June 1912, and Flexner 1 in March 1912. The remainder are laboratory strains of unknown age.

In the above table B. Strong shows agglutination in the highest dilution of serum, the greatest number of cases being in the 1—30 column. The B. Y. His and the Flexner strains come next, the majority of cases showing agglutination in a dilution of 1—10.

The Shiga group (the most toxic group) comes last of all with a maximum at 1—5.

B. Flexner 1 (the strain of known age) and B. Y. His approach more nearly the Shiga than the Flexner type of reaction.

TABLE III.

Showing the agglutinating power of the serum of a population who state that they have *never* suffered from dysentery when tested with a series of strains of B. dysenteriæ.

Organisms.	Serum dilutions with number of cases under each.													Total No. of cases.
	—5	+5	10	15	20	25	30	40	80	100	120	140	160	
B. Shiga, 1 ...	26	41	15	7	4	1	1	0	1	0	0	0	0	96
„ 2 ...	24	28	17	3	1	1	2	1	0	0	0	0	0	77
„ 3 ...	22	26	18	4	3	2	1	1	0	0	0	0	0	77
B. Flexner, 1 ...	21	28	17	22	14	2	2	0	0	0	0	0	0	106
„ 2 ...	0	21	28	24	14	8	9	1	1	0	0	0	0	106
„ 3 ...	1	24	22	23	13	7	13	1	1	0	0	0	0	105
B. Strong ...	4	2	3	8	6	9	26	12	7	0	1	0	0	78
B. Y. His ...	12	21	17	12	9	6	1	0	0	0	0	0	0	78

TABLE IV.

Showing the aggultinating power of the serum taken from a population who give a history of having suffered from dysentery *within the past twelve months* when tested with a series of strains of B. dysenteriæ.

Organisms.	Serum dilutions with number of cases under each.													Total No. of cases.
	—5	+5	10	15	20	25	30	40	80	100	120	140	160	
B. Shiga, 1 ...	11	11	4	2	5	1	0	0	0	0	0	0	0	34
„ 2 ...	9	8	3	0	1	2	0	0	0	0	0	0	0	23
„ 3 ...	7	6	6	0	0	1	2	0	0	0	0	0	0	22
B. Flexner, 1 ...	11	6	7	3	4	0	3	3	0	0	0	0	0	37
„ 2 ...	1	7	7	6	3	4	5	2	1	1	0	0	0	37
„ 3 ...	2	5	9	9	5	1	3	4	0	0	0	0	0	37
B. Strong ...	0	1	1	2	3	2	4	6	3	0	0	0	1	23
B. Y. His ...	6	4	3	3	4	2	1	0	0	0	0	0	0	23

TABLE V.

Showing the agglutinating power of the serum of a series of cases who give a history of having last suffered from dysentery *over twelve months ago* when tested with a series of strains of B. dysenteriæ.

Organisms.	Serum dilutions with number of cases under each.													Total No. of cases.
	−5	+5	10	15	20	25	30	40	80	100	120	140	160	
B. Shiga, 1 ..	8	9	6	1	1	0	1	1	0	0	0	0	0	27
„ 2 ...	7	7	4	0	0	0	0	0	0	0	0	0	0	18
„ 3 ...	7	6	4	1	0	0	0	0	0	0	0	0	0	18
B. Flexner, 1 ...	5	9	4	4	2	1	0	1	1	0	0	0	0	27
„ 2 ...	1	4	8	6	3	3	0	1	1	0	0	0	0	27
„ 3 ...	2	3	9	3	5	2	1	1	1	0	0	0	0	27
B. Strong ...	1	1	0	4	3	1	4	2	1	0	0	0	1	18
B. Y. His ...	3	5	4	1	4	1	0	0	0	0	0	0	0	18

Explanation of Tables III, IV and V.—The above three tables show the results obtained when the population given in Table II is divided up according as to whether the individuals give a negative (Table III) or a positive history of dysentery and the latter are again divided into those who state that they have suffered from dysentery within the last twelve months (Table IV) and those whose last attack was over a year ago (Table V).

An examination of the figures will show that there is no appreciable difference between the results obtained in any of the three tables (Tables III, IV and V) when they are compared with each other or when they each are compared with Table II.

The correlation, therefore, between a history of dysentery and a corresponding serum agglutination is very slight. Those who state that they have never suffered from dysentery show very much the same agglutination reactions as those with a history of the disease. This may be due, of course, to want of recentness of attack, comparative absence of bacillary dysentery among the dysenteries, or inutility of the reaction as a diagnostic procedure.

Now, before we leave this subject of serum reactions, we may refer to the usual method of differentiation of bacillary dysentery.

Starting with a known organism, say a Shiga-Kruse variety, we make from it a high titre serum. Then we agree that if a given case of dysentery is true bacillary dysentery the bacilli obtained in culture from the dejections will agglutinate with that serum or one similarly prepared from some other known strain. The argument is, however, somewhat weak. It depends for its validity on our assumption that the strains of dysentery in our possession and to which we have obtained high titre sera are the only possible varieties. We examine the bacteria isolated and find an agglutinating colony. This is sufficient to lead us to give up further search in the belief that we have found the true cause. But the very method of setting about the search represents a begging of the question of the possible co-existence of variations amongst the organisms present alongside that which gives the required reaction.

The test almost certainly implies the rejection of all other organisms but this one which is then put through tests such as absence of motility, fermentation reactions and indol production.

It is not to be wondered at that with such a technique we should be able to confirm previous findings as to special types of organisms. Failing to obtain such a reaction we think ourselves justified in classifying the dysentery as due to another cause than the bacillus. If at the same time amœbæ are found the disease is very likely to be called amœbic dysentery. Other criteria, such as the improvement under the use of anti-dysenteric serum or a drug such as emetine, are also utilised by the diagnostician.

One problem of bacillary dysentery, then, would seem to be worthy of attack with some other starting point than that of the usual method of differentiation of organisms into dysenteric and non-dysenteric by means of high titre sera.

Our idea is that we should return to what must have been the standpoint of the earlier investigators into bacillary dysentery. We might take as potential criteria simply (1) the presence of clinical dysentery, (2) the determination of the most prevalent organism in the characteristic dejections, (3) specific reaction on the part of the patient's own serum to one or other of the organisms isolated from his dejecta. Our tests would be applied to these organisms, not with reference to any opinions on the subject or to any previous description of the true type. We would endeavour, as must have been done originally, simply to find out which, if any, of the organisms isolated may be regarded as the true cause.

For this purpose we must have some sort of tests over and above that of prevalence.

We would suggest an investigation which would include—

(1) An application of serum tests (particularly the patient's own serum and not merely a high titre serum). Such an investigation would take the form of a determination of the possible utility of agglutination, bacteriolysis, deviation of complement, opsonic index and similar tests.

(2) The use of the isolated "most prevalent or suspected organism" in cutaneous or ophthalmic reaction.

(3) An endeavour to complete the proof of causation by inoculation into animals with reproduction of the disease or symptoms of the disease.

If we can satisfy ourselves as to significant differences obtained with such an organism in cases of clinical dysentery, as contrasted with those obtained in healthy individuals or in those suffering from other diseases altogether than dysentery, then we may proceed to compare it with the known strains of dysentery organisms in respect of reaction to high titre sera, sugar fermentations, etc. The procedure we have outlined is a position of non-acceptance of any view as to what is or is not the specific germ of dysentery. Its only assumption is that a bacterium of some kind is the cause of some dysenteries and we hope, by this way of attacking the subject, to be able to obtain a measure of the variability of the organism as regards the characters usually taken as differential. But let it be noted that in this investigation we have as our starting point simply the syndrome of dysentery. Our investigation into the bacteria of dysentery is to be conducted not only without bias from views as to which tpye of organism is the true dysentery bacillus but also without any bias due to our discovery in such cases of any amœbæ or of the amœbæ said to be pathogenic.

This search would be conducted not merely at the onset of the disease but also at intervals in its course and even after cure.

We should, likewise, even in cases exhibiting the characteristics of an amœbic dysentery, apply tests with the known types of bacilli as well as with other organisms isolated, just as if there were no probabilities in favour of the suggested causation.

Our insistence on these points is due to an ill defined feeling, that the controls of the work already done have not been all sufficient, or at least are not given in a form which carries absolute conviction.

The suspicion that causation is, perhaps, not fully established is perhaps greater with reference to amœbic dysentery than in the bacillary form. When

we are confronted with two schools of thought one of which (Schaudinn's) maintains that the amœbæ of amœbic dysentery are true parasites and are incapable of cultivation whilst the other (that of Musgrave and Clegg) assures us that there is at present no means of differentiating pathogenic amœbæ from the free living forms (capable of cultivation) found in air, water and intestinal contents, we may well be pardoned a desire to see further investigation instituted into the matter.

Such an investigation would inquire into the presence of bacillary reactions in amœbic cases, the proportion of cases of other than bacillary and amœbic causation, the presence of bacilli or amœbæ in these "other cause" cases, the occurrence of mixed amœbic and bacillary dysentery, the superposition of amœbic upon bacillary dysentery and *vice versâ*, and the course of events leading up to the chronic forms of dysentery both amœbic and bacillary. It would require the strictest control in the matter of finding bacillary reactions, presence of amœbæ, etc., with a similar investigation into a sufficiently large number of persons in a healthy state or suffering from other diseases altogether. Experimental work on animals with amœbæ of culture and amœbæ of stools, bacilli of dysentery and bacilli not conforming to the type organisms would have to be conducted. Cases of liver abcess would be especially investigated both for amœbic and bacillary findings and such cases would be specially tested for bacillary reactions to autogenous organisms and to the type forms.

The careful investigation into the occurrence of intermediate forms between the various typical amœbæ (number of cysts, colour, character of vacuoles, pseudopodia, etc.,) would be very carefully gone into by recording *seriatim* all characters of all organisms obtained in purely random samples of material.

Proof or disproof of statements as to cultivation of amœbæ using proper controls to meet the objections raised to previous technique would be attempted. In fact, whereas previous investigation has gained its experience of possibilities and probabilities in the course of the investigation, such a one as we suggest would begin *ab initio* as if the ground had not already been traversed, but as far as possible with no bias to one or other opinion. The experience of others would be with us whilst we discarded all positive findings until such a time as they are refound.

So much for the problem of causation.

Treatment.—Here such confusion as exists with regard to benefit obtained is due to the absence of definitive treatment. One remedy is used in conjunction with others and the benefit claimed, not for the combination, but for the single medicament. In any inquiry into the merits of well known remedies these should be used singly or, upon combination, the result should be recorded as due to the combination.

We have such remedies as Magnesium Sulphate, Emetine, Anti-dysenteric serum, native remedies such as *Isaphgul* and *Kurchee* to consider. Of these Magnesium Sulphate and Anti-dysenteric serum are of avail in bacillary dysentery whilst Emetine is of value in the amœbic form. Thus the form of treatment is evidently largely dependent on the discovery of the causation of the disease.

But if we turn to statistics of causation we are confronted with the fact that the cause of a very large proportion of cases of dysentery is undiagnosed and possibly cannot be diagnosed. Such, at least, is the impression we would gain from a statement made by Captain Wells in his Scientific Memoir * that out of 268 cases of dysentery, 14 were diagnosed bacillary and in 52 motile amœbæ were found microscopically.

It might then be worth while to investigate the action of the various remedies on a very simple basis, namely their administration for the condition which we know as the syndrome of dysentery. Our facts as to the effect would first be collected on this foundation alone with strict adherence to an alternative or serial method of administration. We could then proceed to test and cross test the effects obtained where causation was taken into account.

As strict controls as possible would be provided for all the trials and thus finally we might be able to make definite pronouncement as to the selectiveness of action of the advocated remedies.

* Scientific Memoirs No. 52, 1912.

We have made no mention here of the use of dysentery vaccine in treatment. This method of treatment was originated and carried out in this country with good results by Major Forster, I.M.S.

We have not done so because this remedy is excluded from use in the acute case and it seems more natural to consider its application in prophylaxis than in treatment. Prevention of relapse, maintenance of health would seem to be more important functions of this remedy than the actual cure, which doubtless it can also bring about in a certain class of case.

Prophylaxis.—Under this heading we may consider for the sake of argument four measures :— (1) Vaccination, (2) Purgation, (3) Segregation, (4) Sanitation.

(*1*) *Vaccination.*—We have referred to the use of vaccines therapeutically in cases which have passed the acute stage of the disease.

Our present theme is, however, the use of vaccines prophylactically for the prevention of relapses. Vaccines have been extensively used for this purpose. It would seem obvious that a vaccine, if its action is a specific one, would be of service only in cases of bacillary dysentery. It seems to us, however, that in an investigation into the limits of usefulness of vaccines we should have to apply the treatment first universally in all cases of clinical dysentery. No other remedies would have to be used along with the vaccine. In this way we should obtain results of a general kind and then the further test might be made as to the much greater benefit which we might expect to obtain when a proof of causation by the bacilli of dysentery had been substantiated. The problem would in fact be very much the same as that with which we were confronted in comparing the value of, say, Magnesium Sulphate in the treatment of dysentery in general, with its value in bacillary dysentery.

Other problems such as the relationship of the type of vaccine used to the type of organism found might be attacked if opportunity and time allowed.

(*2*) *Purgation.*—Captain McKendrick, I.M.S., conceived the idea that as Magnesium Sulphate was often successful in the cure of dysentery, the purgative effect of this drug might have an important connection with the cure and so was led to extend the idea to prophylaxis.

Prisoners were placed at regular intervals (once weekly) on a laxative diet with the definite idea of the prevention of dysentery.

The method has also been applied by using Magnesium Sulphate itself in place of a dietetic laxative. The results were good and the method should be given a trial in controlled fashion.

(*3*) *Segregation.*—We may conceive of a situation in which mild cases of dysentery were treated merely as out-patients without admission to hospital or of a situation in which every case, however mild, was admitted to hospital until all symptoms had disappeared and possibly till some time after that. In the one case we should be passing back into the general population individuals who might be infective for others, whilst in the second we should be *segregating* cases in the presumably most definitely infective stage.

It is possible that in applying some of our prophylactic remedies, some of the credit claimed for the medicament used should go to the associated procedure—segregation in hospital. We should, therefore, endeavour by separate and controlled test to see what the effect of segregation alone and in conjunction may be.

Other methods of segregation than admission to hospital are also possible, but our insistence here is on the fact that admission to hospital is of the nature of segregation and should take its due place amongst measures calculated to reduce incidence and mortality.

(*4*) *Sanitation.*—It is likely, with a disease attacking the lower bowel and whose cause is almost certainly to be found in the dejecta that the sanitation which attends to the disposal of those dejecta, the prevention of contamination of food and water, will be effective in limiting the spread of the disease. When

attention is specially concentrated upon the ravages of dysentery, special investigation is made into the causation and measures adopted for the prevention. Sanitation is invariably one of the points looked into. Here again some of the credit for the results obtained and medication may have to go to the head of sanitation and it would be well in future investigations to take this into account.

But the question of sanitation—the prevention of the distribution of infective dejecta over food and into water raises the further question of the role of the "carrier" in dysentery.

The carrier.—With the advent of the conception of the role of the "carrier" as the real cause for the upkeep of diseased conditions entirely new views may be said to have been adopted on the subject of disease prevention. The idea had its inception with reference to Typhoid fever but has been widely extended. The preventive medicine of the future is likely to look not so exclusively to the danger of the acutely infected individual to his fellows but to the individual, or as the case may be animal, that represents the reservoir from which, as source, outbreaks of disease occur.

The carrier, then, whether as an individual just recovering from the disease or one in more or less permanent ill health, suggests a problem for the consideration of the investigator of dysentery. Proposals for dealing with this class of case whether in restricted populations such as those of regiment, jail or asylum, or in the general population should be forthcoming in connection with any investigation of the subject. Detection, segregation, vigorous and continuous treatment of the dysentery carrier should produce good results on the transmission rate of dysentery and therefore upon incidence and mortality rates. Sanitation should produce its effect by diminishing the possibilities of distribution of infection from carriers. The investigation would be directed to determining whether the term carrier could be applied to individuals in apparent good health as well as to those who, having contracted dysentery, never cease to suffer from recurrence of the disease in some shape or form.

Our proposals may now be formulated with reference to the special points raised in this discussion.

These proposals form the constructive side to the negative criticism which we have offered upon current ideas. We fully recognise too that these criticisms partake of the arm chair variety and accordingly we present them with all due and corresponding humility.

Proposals.—(1) That an investigation is desirable into the causation, treatment, and prevention of dysentery.

(2) That such an investigation being of a very wide scope requires careful preliminary preparation on the part of the investigators.

(3) That, in order that results should be forthcoming within a reasonable time and be as comprehensive as possible, it is clearly necessary to have a considerable number of trained hands engaged in the work.

(4) That the enquiry should start with known facts only.

All superstructure should be discarded and only foundations accepted. The superstructure to be reared would be in accordance with experience won, but would be of a form of much greater stability than any previously raised—at least such would be our hope.

INTERIM REPORT ON THE CAUSES OF DIARRHŒA IN POONA.

BY

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Since April of the present year, at the request of the Government of Bombay, an enquiry into the causes of diarrhœa in Poona has been conducted from the Parel Laboratory. Poona, a city of 117,256 inhabitants with an adjoining suburban Municipality and a large Cantonment comprising in all 156,693 persons, is situated on the right bank of the Mulla Mutha River as the latter flows across the Deccan plateau 1,850 ft. above the sea-level. The ancient seat of the Peshwas, its importance in point of size and the climate which it enjoys during the monsoon have made it at this season the headquarters of the local Government.

2. In spite, however, of favourable climatic conditions Poona for some years has had a reputation for unhealthiness based chiefly on the prevalence of enteric fever and latterly of diarrhœa. No reliable records exist to enable us to test from year to year the health of the station in relation to the diarrhœa and the reasons for this are that comparatively little is known of the real extent of the disease in the city itself and that in the suburban municipality and in the cantonment the diarrhœa among a population, composed largely of European adults, though it may cause much inconvenience is not in individual cases so severe as to materially affect the admissions to hospitals or the mortality returns. Nevertheless, so familiar has monsoon diarrhœa become with the members of the European community that for some years the complaint has been known by the fanciful name of "Poonaitis."

3. A few years ago the diarrhœas that form the subject of this enquiry were overshadowed in importance by the prevalence of enteric fever; but, in the absence of definite records, several individuals are of the opinion that diarrhœa is no new complaint in Poona and it is of interest that, in June 1825, Bishop Heber, arriving at the break of the monsoon, was confined to bed for several days with a mild attack of dysentery.

4. Preliminary enquiries elicited great differences in opinion as to what constituted the symptoms that were characteristic of the local diarrhœa. The acting Civil Surgeon and the more senior Medical Officers described the Poona diarrhœa as chronic, essentially resistant to treatment, with large loose light coloured and frequently frothy motions, free from blood and mucus. Abdominal pain was usually absent or, if present, only of a vague character. On the other hand, the Staff Surgeon, the Surgeon to His Excellency the Governor and several of the regimental doctors laid stress on the suddenness with which an attack usually began, the griping pains in the abdomen immediately preceding an urgent desire to go to stool and the copious watery motions which characterized an initial attack.

Of the latter type were the majority of the cases brought to our notice during the past season and the brief notes on the morning sick in the records at the Station Hospital would indicate that this type was equally frequent, at any rate among soldiers, in 1911.

5. Moreover, previous to this investigation, Captain Stanger-Leathes, I. M. S., had written a valuable clinical account[a] of the illness based on 2 years' experience as Staff Surgeon and in it accurately described the main features of the diarrhœas that have prevailed this year. His report was dated January 1st, 1912, and he thus described the symptoms.

6. "THE COMMON ORDINARY FORM commences suddenly with violent watery diarrhœa and vomiting, loss of appetite, a feeling of great malaise, headache, griping and discomfort in the abdomen, over which on palpation, there is a good deal of tenderness and gurgling. The tongue is dirty, and there is generally some pyrexia at the commencement. The motions are generally very frequent. At first, two or three in one hour, they gradually abate under treatment, and cease in two to three days leaving the patient very weak and debilitated.

OTHER FORMS. "(1) *Subacute.*—The patient feels weak and depressed, has no appetite, has a dirty tongue, suffers from dyspepsia and has three or four loose motions a day usually after meals and in the early morning; very often he is not ill enough to lie up. This form is often very persistent, and if not treated at once by diet and rest, it is very likely to become chronic.

"(2) *Choleraic form.*—This is not very common, the patient has most of the symptoms of cholera; the onset is sudden with very acute griping pains, loose watery motions every few minutes, incessant vomiting, cramps in the legs, and abdomen, sub-normal temperature and collapse.

"(3) *Chronic form.*—After one or two acute attacks or in continuance of a sub-acute attack the patient has a tendency to diarrhœa and dyspepsia whenever any but a very simple diet is taken, this may continue for months and generally necessitates a change from the station, when they quickly get well.

"(4) *Form in infants.*—It is very common and usually very severe. Frequent green slimy motions are present with occasional streaks of blood and very often high pyrexia vomiting and sometimes convulsions. The diarrhœa is very persistent and occasionally ends in death from wasting or convulsions.

"RELAPSES of the ordinary form are common, but very often people seem to get acclimatized after long residence in Poona."

7. An analysis of one hundred and seven cases during the past season shows that, excluding twelve cases which we have good reason to believe were true cholera and in no way related to the other cases under consideration, fifty-nine cases of diarrhœa brought to our notice were of the acute watery type. Their stools were alkaline and were so fluid that on standing they quickly separated into two layers. Microscopically they consisted mostly of vegetable debris and bacteria; catarrhal cells and leucocytes were absent or few in number and red blood corpuscles were not seen. Of the remainder, three occurred in infants, two were sub-acute diarrhœas, six were chronic diarrhœas, one was a case diagnosed as sprue and the remaining twenty-four were more or less frankly dysenteric; of these twenty were acute, and four were sub-acute. Dysentery diagnosed as such by the presence of mucus and blood in sufficient quantity to be visible to the naked eye, contributed one-third of the cases.

8. To explain the causation of the common diarrhœas some, referring to the acute diarrhœal and choleraic types and to an extensive outbreak of diarrhœa that followed a Club luncheon three years ago, dwelt on the probability of ptomaine poisoning and the opportunities that flies have of infecting cold foods with organisms of putrefaction.

(a) Report to the P. M. O. (Poona) Division.

Others and among them certain sufferers from subacute and chronic forms of diarrhœa blamed the drinking water the mineral constituents of which they believed increased with the onset of the rains.

Others, interested in infantile diarrhœas, considered that milk from cattle fed on new pasture played some part.

A former Civil Surgeon and the present acting Civil Surgeon drew attention to the presence of flagellates in the stools.

Not a few held that the disease must be due to an unknown bacterium transmitted from fæces to the food by flies and in the opinion of several, chills were the factor of chief importance.

9. No group of cases has this year suggested a diagnosis of ptomaine poisoning.

An exhaustive quantitative analysis[b] of daily samples of the drinking water has shown conclusively that in its soluble mineral constituents there is no appreciable variation with the onset of the rains.

Experiments with a small number of selected individuals have shown that immunity to diarrhœa is not secured by taking milk from specially stall-fed cattle.

10. Flagellates have been found in eleven cases. Of these in ten cases the organism was almost certainly the Trichmonas and in one it resembled Lamblia intestinalis. The flagellates occurred in all types of the disease being found seven times in acute watery diarrhœas, twice in chronic diarrhœas. once in acute dysentery and once in true cholera.

11. In the hope that the distribution of flies might be correlated with that of diarrhœa flies have been collected on fly papers daily from thirty houses distributed over Poona Municipality and Cantonments and Captain Lucas, R. A. M. C., kindly arranged for a daily collection at Ganeshkhind. Observations in the city were a failure as the papers were invariably tampered with.

A correlation could not be established as so many cases of diarrhœa failed to report themselves that our cases illustrate the limits of a certain Surgeon's practice rather than the incidence of diarrhœa. In a selected regiment, however, where most of the cases were reported, the correlation is not striking.

12. The ample attractions of a dirty kitchen always account for more flies in a house than the proximity of stables or cattle yards. Structural improvements such as wired doors and windows are ineffectual if the floors and the tables be greasy. Minor breeding places are fairly numerous, but, contrary to what is usually stated, horse dung and litter play little part in producing flies in Poona. The great breeding places are the filth pits for the manufacture of poudrette and the collections of cow-dung. The conditions at any one place alter from week to week but it is difficult to describe the loathsome appearance of the filth pits when seen in August buzzing with flies and crawling with maggots.

Cow-dung during the rains is collected in heaps and there are many heaps just outside the limits of the City and the Cantonment. Others are surreptitiously kept in court-yards and behind hedges and all, in June, July and August yield ample supplies of fly larvæ. We were early aware of this, but Babu S. K. Senn discovered that droppings and cow-dung cakes were no less fertile breeding places and from one cake selected fortuitously on the 9th September

(*b*) The analysis embraced a quantitative estimation of the calcium, magnesium, sulphates, carbonates and chlorides present in and the alkalinity and the hardness of the tap water.

we obtained 1,139 larvæ of the domestic fly. If flies play a part in the spread of diarrhœa in Poona it may be that they carry the infection to man not from the latrine so often as from their cradle of cowdung.

13. From the clinical accounts of Poona diarrhœa, which were supplied last April, it was impossible to suspect any known organism and in order that a clue might not be lost, it was thought best to avoid a search directed to the isolation of a definite pathogenic bacillus.

A study of the predominant organisms in each stool was suggested to us, but each case of diarrhœa in a small experimental series taken from a regimental hospital seemed to have its own predominant bacillus and a bacillus most numerous on one day might be altogether missed when a second sample was obtained. Even in a fatal case of acute bacillary dysentery an examination of the three most numerous organisms in the fæces during life and again in the intestine after death failed to discover the B. dysenteriæ (Shiga) which existed in the liver in pure culture.

14. A consideration of the whole intestinal flora thus became necessary. Such a study naturally falls into two parts of which the first is a general study of the stools in health and in diarrhœa, to determine the organisms that occur in the special diarrhœa under consideration and the second part is directed to ascertain the relations which one or more specific organisms have to the disease.

In the latter part specialized methods are permissible and indeed imperative if a definite organism is being searched for, but in the general study it is probably best to have a wider knowledge of the general flora and to bear in mind that the number of times any one organism is found may be an under-estimate of its actual prevalence.

15. The examination of healthy stools, if a strictly fair comparison with pathological material is to be made, should proceed side by side with the examination of cases of diarrhœa; but early in our work it was found that a thorough study of the pathological material that came to hand taxed the resources of our staff to the utmost and our capabilities were limited by the number of test tubes a hardworking staff were able to clean and fill.

16. The procedure adopted was to plate portions of the stool, diluted with saline when necessary, on litmus lactose agar standardised to *plus* 10 acidity. Inhibitory agents such as bile-salt or crystal violet were omitted lest with aerial organisms the growth of those specific to the disease might be interfered with.

The details of the technique underwent modification as experience was gained and latterly the rule has been to examine two to ten plates from each sample. Each plate at the end of 24 hours is described and the colonies are classified as minute, small, medium and large; red, blue, clear, translucent or hazy. The approximate proportion of each class is noted as a decimal and from each class of colonies on the plate representatives are picked off and planted on agar.

17. These agar colonies our experience has shown should be replated to ensure purity and therafter they are examined for motility their power to retain Gram's stain, to produce indol and to ferment glucose, lactose, mannite, saccharose, dulcite and glycerine. They are then classified in three large groups. The first group includes organisms which fail to act on all sugars. The second group with sixteen sub-classes of which only seven are important includes all lactose fermenters, and the third group embraces those failing to produce gas in lactose, but giving acid or acid and gas in one or more of the other sugars. Bacilli of the alkaligenes class fall in the first group. Organisms of the coli family fall in the second group, while all known pathogenic intestinal

bacteria are included in the third. The selection of the sugars was made to facilitate the identification of the organisms of typhoid, dysentery and cholera and the known bacilli of enteritis.

18. Over fifteen hundred organisms have been studied, but without at present detailing results as these are of a somewhat academic nature and are being scrutinized and revised we may say, that no organism giving a novel group of sugar reactions has been found to be prevalent in Poona diarrhœas but that Morgan's No. 1 bacillus, the Dysentery bacillus of Shiga and of Flexner and two unnamed fermenters of glucose and mannite and of glucose mannite and saccharose respectively have occurred in forty-seven out of ninety-five cases and that the first three organisms, recognized as being pathogenic to man, have in certain cases agglutinated with the patient's serum.

19. In ninety-five cases of diarrhœa or dysentery an organism corresponding in morphology and in cultural reactions to Morgan's No. 1 bacillus was found sixteen times.

Of these sixteen cases ten had acute watery diarrhœa, five acute dysentery, and in one the patient, reported as a subacute diarrhœa, sent for examination a scyballus stool covered with blood and slime. None of the cases had lasted for more than a few days, but one case which had acute diarrhœa had several relapses and was seen two months later suffering from acute dysentery. In case No. 103 the patient's serum on the ninth day of the disease gave a trace of agglutination in dilutions of 1 in 20 and 1 in 40, and a week later the reaction was well marked in 1 in 80 and showed to a slight degree in 1 in 200.

It is of interest to note that in four cases with blood and mucus in their stools and in two acute diarrhœas Morgan's bacillus was associated with at least one other non-lactose fermenter; while in eight cases of acute diarrhœa and in two cases of dysentery Morgan's No. 1 bacillus was the only non-lactose fermenter found.

20. An organism resembling Shiga's bacillus in morphology and in culture was found in sixteen cases. One case had been returned as cholera, though no cholera spirilla could be isolated from the stool and the patient made a rapid recovery. In seven cases the symptoms were those of acute diarrhœa and no mucus or blood was passed; in four others blood and mucus were present in the stool; in one more the stool was fæcal and contained a considerable amount of slime; in two the stools were semi-solid, and, lastly, there was an ambulant case of dysentery who throughout his attack performed his duties in the barracks.

21. Organisms of the mannite fermenting or Flexner type of the dysentery bacillus were found in ten cases. In two cases the organisms gave an acid reaction in sorbite; the other eight resembled the true Flexner's bacillus. Six were cases of acute diarrhœa; in three others the stools were frankly dysenteric. One case of sub-acute diarrhœa proved on inspection to be passing blood and mucus. Two occurred in infants. The stool of one child gave Flexner's bacillus in pure culture and its serum agglutinated its own organism in a dilution of 1-80.

In no case have the Shiga and Flexner organisms been found together, though other observers have noted this association, but the Flexner bacillus has been found once with Morgan's No. 1.

22. Of the other non-lactose fermenters the two most important groups are those fermenting (*a*) glucose and mannite, type XXVIII, and (*b*) glucose, mannite and saccharose, type XXIX. The first class was found in seven cases and the second also in seven. In two of the twelve cases both organisms were

present. One member of Type XXIX was found once in association with Shiga's bacillus in a case of acute dysentery and one bacillus of Type XXVIII occurred with a Shiga's bacillus in a convalescent case of diarrhœa. There seemed to be no clinical features to differentiate the cases in which these organisms occurred from those in which the better known organisms of the dysentery class were found, but we have not tested the serum reactions.

23. In spite of the kindly interest taken by all Medical Officers the epidemiological statistics that we have obtained are meagre and of doubtful value, but from one household the information was very suggestive.

The stool of a patient suffering from a sharp attack of acute dysentery yielded a strain of the bacillus dysenteriæ (Shiga) which agglutinated with the patient's blood in all dilutions up to 1 in 400. The patient had come from England 10 months previously as governess to two children and since her arrival had had two attacks of diarrhœa and one previous attack of dysentery.

The previous attack of dysentery had occurred about 6 weeks before the present illness, had lasted only a day and a half and had coincided with slight intestinal disturbances in both the children. The present illness had begun two days after the head of the house had been attacked with griping pains and diarrhœa which laid him up for five days. The blood of the latter agglutinated the dysentery bacillus from the governess in a dilution of 1 in 100 and one of the children gave a partial reaction in 1-20.

Of the servants nine either cooked or served the food. The cook, the head butler, the masalchi and the bearer gave no response to the serum test. Two hamals, an elderly table boy and the children's boy gave distinct agglutinations in 1 in 20 and the children's boy a partial response in 1 in 50. Most of the servants had been for years in the household. Of those giving no serum reaction all declared that they had never had dysentery; statements, however, on which there can be no reliance.

Of the four giving serum reactions one admitted an attack of dysentery five years ago and a second informed us that he was just recovering from an attack and that his wife and child were now ill. There was no evidence to show that either of these servants were the active source of infection, but an enquiry into the household management elicited the information that milk after it had been boiled was placed in the charge of the European nurse and that the only servant who had access to it and who alone cleaned the milk vessels was the children's boy who as we have noted was one of the suspected four. The latter supplied one sample stool from which we failed to obtain Shiga's bacillus, but all requests for further samples were denied. Flies in this instance played no part.

24. It now remains to establish by serological methods the identity of the organisms that have been isolated and should the examination of healthy individuals now in hand show that the bacilli of the dysentery group are as rare in health in Poona as in healthy stools in other countries the way will be clear for the more specialized study of these infections which, it is hoped, will be carried out next season.

25. Though this is but an interim report it is our pleasant duty to acknowledge the help we have received from the Medical Officers both Civil and Military in Poona and to thank them for their encouraging interest in our work and for the valuable suggestions we have from time to time received.

POONA, *9th October* 1912.

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ALL-INDIA SANITARY CONFERENCE,—MADRAS,—NOVEMBER 1912.

Prevention of Tuberculosis in Madras.

By

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At the last Conference in Bombay, there was a discussion on the spread of tuberculosis in Calcutta. But as the disease is not confined to Calcutta and is fairly widespread in various parts of India, I invite further discussion on the subject. Tuberculosis is a very common disease and is distributed over the whole civilised world. No race is exempt from the ravages of consumption and no country is exempt. No single climate grants immunity from tuberculosis. It is prevalent in all latitudes and in all altitudes. It is known to occur in all regions whether dry or moist, high or low, warm or cold. The comparative infrequency at high altitudes is easily explained by the sparseness of the population. The frequency of the disease is determined more by overcrowding, poverty, occupation, and neglect of the laws of health, than by geographical situation. It has not drawn the attention it deserves in this country.

The universal distribution and the great prevalence of the disease in various parts of India, the high rate of mortality from it, the curability of the disease in the earlier stages if recognised early, the easy preventability of the spread of infection in the early stages (before the conversion of the closed cases into open cases by insufficient or faulty treatment), the great infectivity of the disease in the later stages and the absolute hopelessness in the later stages of the disease, all point to the necessity for the adoption of suitable measures towards the cure, limitation, and control of this disease.

For a long time medical men in this country have been unwilling to recognise the prevalence of typhoid fever and tuberculosis amongst the native population of India. But during the last twenty years medical men have been gradually convinced of the existence of both these diseases amongst the Indians. Doctor Turner, Captain Stephens, Colonel Roberts, Doctor Chatterjee, Major Calvert, Doctor Harris and Colonel Rogers, have all drawn attention in recent times to the great prevalence of tuberculosis in such widely separated places as Bombay, Darjeeling, Bengal and the Central Provinces. The study of the *post-mortem* records in the Calcutta Medical College Hospital by Major Rogers brought out the startling fact that 25 per cent of the bodies examined *post-mortem* showed signs of active or latent tuberculosis. Here in Madras, the disease is fairly common and is distributed over the whole Presidency. The percentage of bodies in which tubercular lesions were found *post mortem* in the General Hospital, Madras, during the last five years is given below.

	Percentage of the bodies examined.
1907	27·7
1908	8·4
1909	11·5
1910	9·0
1911	11·2
The average for the last five years is	13·56 per cent.

The returns of the Surgeon-General, Madras, for cases treated for tuberculosis in the hospitals and dispensaries in this Presidency show an increase year after year. Is the increase really due to an actual increase in the prevalence

of this disease, or due to greater and greater precision in diagnosis? I believe it is due to both. In this country, it is very difficult to obtain accurate returns for the deaths from tuberculosis. The figures available must be far below the actual number of deaths owing to inefficient registration. I hope that some systematic attempt will be made to secure fairly reliable vital statistics for the whole Presidency, when the new regulations for the improvement of the Sanitary Department are given effect to. A desirable and necessary reform in the matter of improvement of the village sanitation will be to group the villages together to form village unions and to see that no village is left out. Every village should form part of some union. All the villages within a radius of 5 or 6 miles may be formed into a union with a Council and a Chairman responsible for the sanitation and the roads of the union villages. If the Chairmen of the various unions and municipalities (and the village headmen in the absence of village unions) be made to submit returns to the Sanitary Commissioner, through the Deputy Sanitary Commissioners, for all births, deaths (under different headings) and cases of infectious diseases, *e.g.*, cholera, small-pox, leprosy, tuberculosis, plague, etc., in their jurisdiction, much valuable information can be obtained and published by the Sanitary Commissioner as regards the distribution and prevalence of the infectious diseases and sources of infection.

In each village, the village headman might be held responsible for the supply of the information. The figures should be verified, if possible, by the Taluk Sanitary Inspectors and the Deputy Inspectors of Vaccination, by personal enquiries during their visits to the villages when on tour. Wilful or negligent errors should be reported for further action to the higher authorities concerned. The establishment of these village unions is a necessary and important factor in the improvement of the sanitation of the rural areas in this Presidency.

At present we have to be satisfied with the returns of the Surgeon-General, if we want to gather any information as to the prevalence and spread of tuberculosis in this Presidency. The Surgeon-General's figures refer only to the hospital-attending sick people who constitute only a minority of the population, even in localities where hospitals are situated. And there are many places where there are no hospitals in the neighbourhood. While the people in this Presidency have not yet lost faith in the native methods of treatment in medical cases, the European methods of medical treatment have not yet won the confidence of the public. The hospitals are resorted to more for surgical help than for medical help. The percentage of the people resorting to the hospitals and dispensaries for medical treatment in this Presidency during the year 1910 was about 14 per cent (or 1 in 7). The "morbidity factor", if I may use this expression, might be taken as 7, or in other words, the figures given in the Surgeon-General's report for any particular disease might be multiplied by 7 to give us some rough idea of the prevalence of that disease. The figures given below and taken from the annual reports of the Surgeon-General, Madras, show clearly that there has been an increase in the returns for tuberculosis during the last 10 years.

Total number of cases of tuberculosis treated.

Year.	Number.
1901	7,458
1902	6,544
1903	6,467
1904	8,543
1905	11,603
1906	8,980
1907	9,252
1908	9,949
1909	10,738
1910	13,990 or nearly 14,000.

This table brings out clearly that the number of cases of tuberculosis treated has risen cent per cent in 10 years A study of the annual report of 1910 brings out prominently the widespread distribution of the disease throughout the Presidency.

If we arrange the districts in order, starting with the district that had the largest number of admissions, Malabar heads the list and is followed by Madras, South Canara, Tanjore, Coimbatore, Madura, Tinnevelly, Nellore, Kurnool and Trichinopoly in the order given. Nilgiris stands fourteenth in the list and has 10 districts below it, disproving thereby the popular erroneous belief that tuberculosis is not found in elevated regions. Bellary, which is very often recommended as an ideal place of residence for comsumptives by medical men who have never been there, stands 16th in he list (13th in the list if the average be taken for the last 10 years) and has eight districts below it. The reports of the Surgeon General record 1,249 admissions during 1910 for tuberculosis in the hospitals and dispensaries of the city of Madras. At least an equal number of cases may be supposed to be under treatment by private practitioners outside the hospitals. Besides, many cases of tuberculosis are being treated in Madras as malarial fever, bronchitis and influenza. For some time past I have been in the habit of examining the relatives of the consumptive patients who come for treatment, and the impression left on me as the result of such examination is that for every consumptive who applies for treatment, there are at least two more cases in the house in an earlier stage of the disease and not undergoing treatment. A very modest estimate of the number of people affected with tuberculosis in this city will be about 8,000. I am sure that a systematic search would reveal the presence of a far greater number of cases. If we multiply the admissions recorded in the Surgeon-General's report, for Madras, for 1910, namely, 1,249, by seven which I called "the morbidity factor" we get 8,743 (not far from my estimate).

During the year 1911, there have been 760 deaths from tuberculosis alone in this city. The actual figures are likely to be certainly higher as the causes of death are not likely to be correctly entered when the treatment before death has been in the hands of Vythians and Hakeems who thrive very largely in this city. Even in the case of patients treated by qualified men the diagnosis of tuberculosis is often missed. Many cases of tuberculosis pass muster under "Respiratory diseases other than Phthisis", "Pyrexia of unknown origin" and "other fevers than small-pox, etc." I hope medical men in this country would in future make some attempt to investigate the cases of "Pyrexia of unknown origin" and classify them under proper headings. During the year 1910, 71,599 cases have been returned as "Pyrexia of unknown origin" in the civil dispensaries of this Presidency, and 8,653 cases have been returned as such in the European and Indian armies in India. The fact that 1,064 deaths (5 per cent of all deaths) are recorded during 1911-12 in the city of Madras alone under the heading "Pyrexia of unknown origin", should make us ask for an early investigation of the nature of this fever. In the annual report of the Sanitary Commissioner with the Government of India for 1910, it is stated that this group is really made up of 5 types of disease:—

1. Probable malarial cases in which no parasites can be found.
2. One-day fever due to chills, over-fatigue (I have seen three tubercular patients suffering from fever for one or two days as the result of over-fatigue. Burton Fanning of the Kelling Sanatorium, Norfolk refers to a similar experience in 10 per cent of his cases).
3. Sand-fly fever lasting for three days.
4. The seven-day fever of Rogers, or Dengue.
5. Serious cases of long continued fever which sometimes terminate fatally, the diagnosis being obscure throughout the course of illness.

To this list I beg to add as a sixth type, cases that are really tubercular but in which the diagnosis is missed. I would not exclude tuberculosis unless Von Pirquet's test be negative.

Accurate mortality figures will be available in this city only if the municipality insist on the production of death certificates by qualified medical men or *post-mortem* reports in the absence of such certificates, before the disposal of the dead bodies. As *post-mortem* examinations will be distasteful to the people, such a regulation would drive the people from the quacks and make them resort to the qualified medical men, who will give better treatment to the patients and more accurate returns to the municipality than is possible at present. Even if the people be unwilling to give up the quacks, the qualified medical men will be called in at the last moment at least, for the purpose of obtaining a medical certificate. Any way, the municipality will secure accurate mortality figures.

Of all the mortality figures available I consider the figures contained in the annual reports of the Oriental Insurance Company the most reliable as the lives are healthy to start with and as death certificates are invariably insisted upon. During 1911, out of 690 deaths among the policy holders, 62 deaths were reported to be due to tuberculosis. The rate of mortality from tuberculosis (this is for the whole of India) is thus 1 in 11 or 9 per cent of all deaths. It is not far from the rate for England and Wales and the rate for the United States of America, *viz.*, 11 per cent.

The rate for the whole civilised world according to Dr. Kidd is 7½ per cent or about 1 in 13. The fact that 9 per cent of the deaths amongst persons who had been originally certified to be free from disease is due to tuberculosis, is by itself enough to show that the disease is fairly common in this country.

The true infectious nature of the disease was firmly established by the classical experiments of Koch in 1882. Subsequently the studies of Cornet indicated the probable mode of infection to be from the dust of infected rooms and the surroundings of the consumptives. Later on Fugge brought forth strong experimental evidence of "Moist droplet infection" during hard coughing, speaking and sneezing. Recently Behring has suggested that bovine and human tuberculosis are the same and that in most cases the infection is derived from milk during infancy. Koch's belief was that bovine tubercle bacilli were not pathogenic to man; but recent researches in England, America and Germany show that the bovine bacilli are certainly pathogenic to man in some cases, though in the vast majority of cases the infection is derived from the human bacilli in the infected sputum, whether the infection be through the respiratory tract or the alimentary tract. Professor Delepine pointed out at the Tuberculosis Conference held in Manchester last June that 20 per cent of fatal tuberculosis in children was due to infection through the alimentary canal, probably food, and that in 22 per cent of the adults dying of tuberculosis the bacilli discovered were bovine; his conclusions were at variance with the conclusions of Koch. The conclusions arrived at by the International Congress of Tuberculosis held at Rome this year were:—(1) prophylaxis of tuberculosis must be principally directed against the suppression of infection from man and principally in the family: (2) though the infection of man from bovine sources is of less frequency, it is necessary to maintain prophylactic measures against infection of bovine origin. Whether we agree or not with Koch in his conclusions as regards infection of man with bovine tubercle bacilli, infection from cattle through milk and meat may be practically ignored in this country as meat and milk are not taken raw. Man is the chief source of danger for man; and the sputum of the consumptive suffering from open tuberculosis plays the most important part in the dissemination of the bacilli. The infective sputum, dust and the infective droplets from the mouth, are the main sources of infection. It must not be forgotten however that the common domestic flies are active agents in infecting food stuffs with tubercle bacilli. Tuberculosis is so largely a family disease that the importance of family infection can hardly be exaggerated. The opportunities for infection are innumerable not only in the houses of the poor but also those of the wealthier classes in this country, partly on account of bad ventilation and lighting, partly to delay in the recognition of the disease both by the patients and the medical men, and partly on account of the ignorance of the infectious nature of the disease. In the earlier stages, tuberculosis is often mistaken for influenza, bronchitis, malarial fever, etc. It is of the greatest importance to recognise cases of tuberculosis

in the earliest possible stage. In the early stages there is no possibility of transmitting the disease to others. The consumptive becomes a source of infection only when he begins to expectorate. It is exceptional to have actual illness in the earlier stages of tuberculosis unless this follows pleurisy, pneumonia, measles, typhoid fever, when the chief evidence will be incomplete recovery from the illness. In this connection may be noted the interesting observation of Colonel Roberts that some of the cases of typhoid fever in which one does not obtain a positive Widal reaction, are really early cases of tuberculosis, and that they react to the tuberculin tests. The early consumptive does not seek for medical advice as a rule. He has to be sought out, particularly in the houses of late consumptives who come in for treatment. Persistent debility, persistent dyspepsia, tardy convalescence from acute disorders, persistent pallor or anæmia, persistent cough, low fever, progressive loss of weight, frequent attacks of cold, a rise of temperature after meals or after slight exertion, tachycardia, profuse perspiration during examination, hæmoptysis, are all symptoms that should rouse our suspicions and make us investigate the cases thoroughly. The disease usually begins insidiously and is often wrongly diagnosed in this country. In the early stages the disease is commonly diagnosed as malarial fever or bronchitis, or neurasthenia or pyrexia of unknown origin, and treated as such, till the patients become hopeless, infectious consumptives. Even in advanced cases the evening chills of the septic type of tuberculosis are mistaken by some for the rigors of malarial fever, in spite of the absence of the malarial parasites from the blood.

A wrong diagnosis in the early stages *when only the disease can be completely cured*, is detrimental to the interests of the patients as well as of the public. The patient whose condition has not been properly diagnosed rapidly passes on to stages II and III of Turban and begins to expectorate millions of bacilli in his sputum. His condition becomes hopeless and he becomes a source of infection to the public. The wrong diagnosis is not always due to lack of skill on the part of the medical men but to their disbelief in the wide prevalence of this disease in this country. Some of the European medical officers seem to hold the view that tuberculosis of the lungs runs a very rapid course in this country; but I beg to differ from this view. People in this country do not resort to the hospitals and to European doctors in the early stages of the disease. In the very early stages the consumptives neglect the complaint as it does not interfere with their work, and in the later stages they resort to the so-called native treatment of the Vythians and Hakeems. When, however, they find they are not improving they resort to the hospitals or consult qualified medical men. By this time the condition becomes absolutely hopeless and the downward progress is very rapid. In the earlier stages many of the tubercular patients are unaware of the fact that they are suffering from fever till it is demonstrated by the thermometer. Many a time have I found a temperature of 100° or 101° in patients coming to me with a history only of cough and anorexia though they denied having any fever. The tuberculin circulating in their body probably acts as a stimulant and makes them unconscious of the existence of the fever. The patients in stage I of Turban refuse to believe that they are ill enough to require treatment. Even in the later stages of consumption (stage II), I very often find it difficult to convince the patients of their serious condition.

The preventive measures may be considered under 3 heads:

I. Those intended to prevent the infection of healthy people.

II. Those intended to prevent the conversion of cases of closed tuberculosis into cases of open tuberculosis which are the real sources of danger to the community.

III. Those intended to prevent the spread of infection from the open cases.

As regards infection from bovine sources the possibility of infection in a small percentage of cases has no doubt been recognised by various investigators in Europe and America. But in this country we may ignore this method of infection for all practical purposes, as milk is invariably boiled and as meat is cooked before eating. We need not trouble ourselves much with the tuberculosis of cattle in this country.

I. Consumption may aptly be termed a house disease. The house is the place where the tuberculous subject often becomes infected; and the house is the place where the advanced consumptive disseminates the bacilli most, and infects everybody near and dear to him. Direct sunlight can kill the bacillus in five or six hours, and diffused sunlight in several days, while proper ventilation greatly facilitates the bactericidal action. The bacillus is a parasite of feeble growth and cannot be grown on the ordinary laboratory media as easily and rapidly as many other pathogenic bacteria. In the words of Dr. Philip "it demands specially favourable conditions. Even after successful inoculation while the feeble parasitic life is struggling to assert itself on the more or less resistant soil, the conditions of environment determine the production of the disease". Airlessness, sunlessness, and foodlessness afford great advantages to the tubercle bacilli and enable them to take root in the system.

The prevention of infection in the case of healthy people is mainly a matter of general sanitation. The avoidance of intimate contact with the consumptive in the house, the segregation of the consumptive in a separate room if he is retained in the house, the disinfection of sputum, clothing, etc. (in fact everything that has come in contact with the consumptive), the disinfection of the room and its contents after the death or removal of the consumptive therefrom; personal cleanliness, household cleanliness, wholesome food, proper ventilation of the houses, prevention of overcrowding, regular exercise, regular habits, temperance in food, drink, sleep, and sexual indulgence, prohibition of spitting on the floor, are all useful and necessary measures in preventing infection. These are all measures that should be carried out by individuals. Other measures that should be carried out by the municipality are opening out of congested areas, removal of end houses in blind lanes, demolition of old narrow houses, widening of narrow dirty streets, efficient conservancy, building of model houses, provision of standard plans for houses of different sizes, provision of parks in crowded localities, *insisting on a minimum breadth of houses* (30 ft. at least if not 40 ft.), the *prohibition of promiscuous spitting in places of public resort, and *above all new building regulations that will give sufficient power to the Health Officers to prevent the erection of insanitary houses.* In his notes put up for the last Conference, Dr. K. C. Bose referred to the care taken to improve the sanitation and the endeavours made to improve the health of the people in the city of Calcutta. But in my note this year I have to regret that the Madras Municipal Commissioners are trying to curtail what little powers are possessed by the Medical Officer of Health in preventing the erection of insanitary, airless and sunless houses in the city. People are able to set at defiance all the fundamental principles of sanitation and do actually build houses in total disregard of such rules. The Medical Officer of Health has had the right till now, to insist on the provision of an open space in the house to $\frac{1}{3}$ the extent of the house. Quite recently the Municipal Commissioners found out that this healthy regulation, devised in the interest of the public health, stood in the way of avaricious capitalists, and moved and carried a proposition in the Council to rescind the bye-law which insists on the provision of $\frac{1}{3}$ open space. I hope the Sanitary Commissioner and the Surgeon-General will have a voice in the matter before it is disposed of.

The rules for the building of new houses must be redrafted, at least in this Presidency, by a Committee of medical men which should include the Sanitary Commissioner and the Surgeon-General. *The minimum width of the houses, the provision of open court yards,* the situation and the minimum size of the courtyards, the provision of windows opening into the outer air, the height of the buildings, the question of drainage, etc., should all be discussed in such a Committee.

The most important and farthest reaching prophylactic measure is the education of the public in matters pertaining to hygiene. This can be done—

(1) by giving instructions in hygiene and school hygiene to teachers during their period of training (of course the instruction must be given by medical men).

* It is not enough to pass bye-laws. They must be enforced. It is very common to see nowadays notices put up in the verandahs of public buildings prohibiting spitting. Such notices are not enough; people should be told where to spit, and fly-proof spittoons should be put up in various public places as in America. Of course it is needless to say that the spittoons require daily disinfection.

(2) By teaching the elements of hygiene in all schools (both Elementary and Secondary).

(3) By the delivery of periodical lectures on elementary hygiene and physiology by the teachers or the medical officers in the neighbourhood, to the parents of the boys in the school buildings (some prominent holidays like the Easter, Christmas, or Pongal holidays being chosen for such purpose).

(4) By the delivery of lectures on sanitation in the vernaculars to the villagers by the Taluq Sanitary Inspectors when on tour.

(5) By the broad-cast distribution of leaflets in the vernaculars on general sanitation and on the prevention of infectious and endemic diseases, *e.g.*, cholera, small-pox, consumption, plague, malaria, filariasis, etc.

(6) By the publication of the same in the vernacular journals of the Presidency from time to time.

In schools short lessons on general hygiene and on the danger of spitting on the floors, and on the danger of licking fingers, pencils, slates, stamps, etc., should be given. The danger of coughing without covering the mouth and the infectivity of the sputum should be clearly pointed out. The public should be educated up to the fact that the sputum is as nasty, as loathsome, and more dangerous than urine and fæces. In dealing with the tuberculosis problem we must not forget that poverty is a very potent cause of tuberculosis. The under feeding due to poverty lowers the vitality of the masses and renders them vulnerable to the attacks of infectious diseases. But the remedy for this predisposing cause lies in the hands of philanthropists.

II. The consumptive will not infect others in the earlier stages of the disease; but the moment he begins to expectorate he becomes a source of infection if certain necessary precautions be not carried out. Therefore it is necessary in the interests of public health to prevent cases of closed early tuberculosis becoming cases of open tuberculosis. Early diagnosis, before the lungs begin to break down, will enable us to cure the consumptives and diminish the sources of infection. The early diagnosis and treatment of the consumptives is really a part of the prophylactic measures. Many medical men refuse to diagnose cases of consumption as pulmonary tuberculosis unless they find tubercule bacilli in the sputum. But by the time the bacilli are found the cases have become incurable and highly infectious. If the physical signs are inconclusive in cases with a suspicious family history or a suspicious previous history of the illness, the tuberculin tests should be applied without hesitation.* Of the tuberculin tests there are five in number :—

1. Calmette's ophthalmic test.
2. Von Pirquet's cutaneous test.
3. Koch's sub-cutaneous test.
4. Morro's test.
5. The intra-dermal test.

Of these tests, Koch's sub-cutaneous test is by far the most reliable and it is of great value inasmuch as it produces focal reaction; but if there be any fever it is not available. As it necessitates rest in bed and two-hourly thermometric readings for about a week, it is not so convenient as the first two tests. Morro's test is not reliable and finds its use chiefly in babies. The intra-dermal test does not seem to have been made use of on any large scale, and I have no

*The Wasserman test, the Cobra venom test, the aglutination test, and the albumen test have all been used only in a small number of cases by some observers. The tests do not seem to be very reliable. The Opsonic test can be depended upon in skilled hands; but it is not easy to carry out and the personal error is said to be great. I have no personal experience of it.

experience of it. Calmette's and Von Pirquet's tests are simple and reliable. They do fail in a small minority of cases, but in the great majority of cases I have found them reliable. In a few cases where they fail to help me in diagnosis, I found Koch's sub-cutaneous test very helpful. Von Pirquet's test as an indicator of active tubercular lesions is less reliable in adults than in children. The significance of Calmette's reaction is very great. There is a great and unjustifiable dread of this test on the part of many medical men. I have never found it prejudicial to the eyes, though the reaction is sometimes severe, particularly in scrofulous cases. I have tested hundreds of eyes with tuberculin, without endangering the eyes in any way. Of course the eyes tested must be healthy and the solution sterile. Calmette's opinion is worth quoting here. "The test was not dangerous, but precautions must be taken to see that the eye was healthy and that the tuberculin was aseptic and pure. Only in 80 cases out of 20,000 or '4 per cent the reaction was severe so that there was less danger in this procedure than in the exposure to X-rays or the puncture of a vein."

A positive Calmette's test even in the absence of any physical signs in the chest, must rouse our suspicions and make us investigate the family history and the history of the illness, and take two-hourly thermometric readings for a few days. If the thermometric readings happen to be normal, Koch's sub-cutaneous test must be immediately carried out.

The treatment of the cases diagnosed as early stages of consumption may be :—

(1) By open air treatment in a sanatorium, or

(2) By tuberculin injections.

My own experience is that all cases of early tuberculosis can be satisfactorily treated by tuberculin injections alone. The best results are obtained in afebrile cases and resting-afebrile cases. I have found the tuberculin injections (with the new tuberculins) very valuable even in mixed infections so long as the evening temperature is under 99·5°. But in resting-febrile cases the stay in a sanatorium is absolutely necessary. A combination of sanatorium and tuberculin treatment must of course give the best results : and such a combined treatment is carried out at present in many of the sanatoria in Europe and America. As sanatoria are costly and can afford only limited accommodation (for a small part of the population) they should be utilised only for cases *which cannot recover by tuberculin injections alone but which can recover by staying in the sanatoria.* Therefore the very early cases and the hopeless cases should not find entrance into the sanatoria. By prompt treatment of the incipient cases of phthisis, whether by sanatorium methods or by tuberculin injections, the patients can be cured and the dissemination of infection can be prevented to a large extent.

III. By far the most important feature in prophylaxis relates to the disposal of the sputum in the sick rooms. Scrupulous care should be taken to avoid contamination of the clothing, bedding, furniture, floor, hands, lips, beard or any portion of the body. The droplet infection can be guarded against by holding a handkerchief before the mouth during coughing and loud talking. Disinfection of the sputum (received in a spittoon), urine, fæces, hands, clothing, etc., are of course essential whether the patient be segregated in his own home or in a hospital or in a sanatorium. As soon as the patient arrives at the infectious stage, it is desirable to remove him to a consumption hospital or sanatorium where he will have to stay for three months at least unless he can be efficiently segregated at home. A few months' stay in the sanatorium would be of great value to him in educating him and teaching him correct sanitary habits. An individual who has lived in a sanatorium or a consumption hospital for two or three months can be depended upon to live in such a way as not to endanger his neighbours.

Notification of all cases of open tuberculosis is highly necessary to enable the health authorities to take proper action. The notification will lead to the supervision of the surroundings, the segregation of the infectious cases and the

search for incipient cases amongst the contacts. Notifications to be effective must be compulsory on the part of medical men, but they must be paid a fee for the notification. It is a mistaken policy to compel medical men to notify without a fee. The Medical Officer of Health will in this way obtain valuable information about the presence and extent of the infection in the City. The Medical Officer of Health or his assistant, after receiving information of the existence of the infectious cases should go over to the houses infected, make an investigation into the sanitary condition of the house and its surroundings, and take steps to improve their sanitary condition. As for the patient he should be advised to go over to the consumption hospital for a few months. If he be reluctant to be removed, the necessary instructions to carry out home segregation and disinfection of sputum, etc., should be given. The other inmates of the house should be advised to get themselves examined by private medical men or in the out-patients department of the consumption hospital. Compulsory segregation may not be practicable at present. We should depend upon persuasion for the present. If willing to be removed the infectious patients should be removed to a special consumption hospital and not to a general hospital as is done at present. The reception of consumption cases in a general hospital will endanger the safety of the other patients whose vitality has already been lowered by other diseases. Madras is in urgent need of a special consumption hospital in the centre of the town, accommodating about 100 patients. The consumption hospital situated in the centre of the town will not be a source of danger to the neighbourhood as the disinfection will be efficiently carried out inside the hospital. The need for such a hospital in Madras is being emphasised year after year by the Senior Medical Officer of the General Hospital. The site of the late Gun Carriage Factory and the Spur Tank will make ideal sites for a consumption hospital. The consumption hospital should have both an out-patient department and an in-patient department. In this hospital the consumptives should be kept at rest and under treatment for two to four weeks. They can subsequently be sorted out into :—

(1) Those who after a brief stay and treatment can be turned over to the out-patient department for treatment with tuberculin injections.

(2) Those who must be turned over to the sanatorium—the resting-febrile cases which are not advanced.

(3) The incurable and hopeless cases which can be kept on till correct sanitary habits are learnt by the consumptives. They can subsequently be discharged, or kept on if there be room. These cases should certainly have a ward of their own.

The out-patient department should be mainly a tuberculin dispensary intended for the tuberculin treatment of the working, afebrile and ambulant-afebrile cases. It is the place where the contacts of the consumptives can be examined and advised. It is also the place where incipient cases not desirous of admission in the hospital can be examined and advised.

People who can neither take their own temperature nor can get friends or relatives to make the necessary observations, *e.g.*, illiterate people, must of course get their injections in the sanatorium. The sanatorium need not necessarily be situated in elevated places or woods. Proximity to the sea is not necessarily objectionable. The British health resorts which have a reputation for the treatment of the consumptives are on or near the sea coast, such as the sanatoria at Bournemouth, Ventnor, Margate, and Torquay and the Cromer sanatorium. A great advantage of the sea-side (also shared by mountain resorts) consists in the recurring breezes which purify the air. Further the proximity to the sea tends to equalise the temperature throughout the year. The belief amongst the Phthisio-therapists at the present day is that no special climate is necessary for a sanatorium, and that the effects of treatment are mainly due to dust-free open air; Herman Weber highly commends warm climates with plenty of sunlight. Dr. Philip of Edinburgh is of opinion that Aero-therapy is a measure of universal applicability in all lands and that there is no climate specially favourable for its practice. He is also of opinion that the sanatorium should be easily accessible. Dr. Galbraith, lately House Physician to Dr. Philip, has written, "Altitude and

climate have been asserted to exert a very marked influence on the course of tuberculosis and yet we meet with sanatoria at all altitudes and in almost all climates with a practically uniform measure of success attending the application of the open air life. * * * * The aim of the treatment is to fit the patient for taking his place in the ranks of the workers and not to produce a crop of exotics to live under artificial conditions of climate or altitude. Thus a person subject to tuberculous disease should be treated under conditions as little artificial in these respects as possible and as nearly alike as practicable, to the conditions under which he will afterwards have to live and work. This view is now generally recognised and all along has been the guiding principle in the Edinburgh system. * * * A public sanatorium must be easy of access from the district which it is intended to serve."

Guindy will be an excellent place for locating a sanatorium for Madras. It is not very near the sea and is conveniently far from Madras. The place is easily accessible and the air is pure. It has also got the advantage of being in the proximity of a small mount, St. Thomas' Mount, which can be ascended by convalescents. It will also be convenient for starting a farm colony later on.

The measures necessary to prevent dissemination of tuberculosis in this country may be summed up as follows :—

(1) Improvement of the general sanitation in towns as well as villages.

(2) Grouping of the villages into village unions for the purpose of securing better village sanitation.

(3) *Improved building regulations in towns as well as villages.*

(4) Education of the public in matters pertaining to general health and infectious diseases.

  (*a*) By means of lectures on sanitation to the public, by Assistant Surgeons, Sub-Assistant Surgeons, School Masters, Sanitary Inspectors, etc.

  (*b*) By teaching hygiene in schools.

  (*c*) By teaching hygiene and school hygiene to the teachers during their period of training.

(5) The early diagnosis and treatment of incipient cases of tuberculosis.

(6) Notification of infectious cases to the health authorities concerned.

(7) Prohibition of indiscriminate spitting.

(8) The provision of consumption hospitals and sanatoria in different parts of the Presidency.

ALL-INDIA SANITARY CONFERENCE—MADRAS—NOVEMBER 1912.

TUBERCULOSIS AND ITS RELATION TO PUBLIC HEALTH.

BY

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This is a very extensive subject and in a short paper one cannot hope to do more than touch the fringe of it. Tuberculosis, its prevalence, mortality, treatment, etc., have all been fully written up by able writers. The books on these subjects are numerous and contain all the information anyone could possibly need for enlightenment. There are anti-tuberculosis societies at work in most civilised countries, and since the late King Edward interested himself in the Anti-Tuberculosis movement, the campaign against this terrible disease has taken on considerable activity in the United Kingdom and Colonies of the British Empire, including India. But of all countries America appears to be the most active in this respect. The object of this paper is to draw attention not so much to what India is doing, as to what India *might* and *should* do for the eradication of this disease which, if the facts were appreciated, is more insidious and more to be dreaded than Plague. If the literature on this subject, with the stern facts on mortality from this disease, could be brought home to the masses in India in simple form, it might result in their valuable co-operation, without which any movement is practically hopeless. There is too much of the tendency in India to look to Government for remedies for all evils. By all means look to Government for support and direction, but the initiation of all measures for the amelioration of public evils should surely be undertaken by the public themselves. It is in this connection that I would invite a consideration of what other countries are doing, as compared with what India is doing, together with a plea for what might be done—prefacing this with a few remarks as to the prevalence of, and mortality from, this disease. From the statistics which are available in most text-books and reports, one reads that the deaths per annum in England, Scotland and Ireland exceed those from other diseases. The number of deaths in England annually is estimated at over 55,000, and in Ireland at about 12,000. It is further estimated that about 500,000 persons in England are suffering from Tuberculosis in some form or other. I also contrast the mortality rates per 1,000 living for the three countries mentioned below in 1876 and 30 years later :—

	1876.	1906.
England	2·9	1·6
Scotland	3·4	2·1
Ireland	2·5	2·7

It is interesting to remark the reduction which has been made in mortality in England and Scotland during the 30 years under review, and the stationary character of the mortality in Ireland. While England and Scotland have been reducing the evils of the disease by better house accommodation for the working class and other measures, Ireland, which like India, is slow in accepting sanitary changes, shows no decrease in mortality. I have quoted the above figures for 1906 from the data given in "Ireland's Crusade against Tuberculosis" by the Countess of Aberdeen in order to bring the case of Ireland more prominently forward for consideration, as India has many points of analogy with conditions of life in Ireland.

Practically speaking the mortality in civilised countries is at least 3 per cent of the population, and 10 per cent of total deaths are due to Tuberculosis. This fact when duly appreciated is terrible to think of. In some countries active measures are being taken to combat the disease by (1) making the disease notifiable, which provides reliable statistics (*a*) for the incidence or prevalence and (*b*) in regard to mortality, (2) establishment of sanatoria for consumptives and segregation; (3) the treatment of consumptives at tuberculin dispensaries; (4) house to house visitation of consumptives by doctors and nurses; (5) distribution of literature warning the public of the contagious nature of Tuberculosis and the precautions to be adopted for the prevention of, and steps to be taken for the cure of the disease, etc. These and other measures are being actively adopted by many countries in their efforts to fight this disease, a commendable campaign which must eventually result in a great reduction in incidence and mortality. So much for other countries. Now let us consider India. I would not like to say India was not a civilised country, but in the matter of statistics we have not attained to any degree of reliability. As long as the registration of deaths is left to the village chowkidar in villages or the registration clerk in towns, we can never hope for reliable statistics in regard to mortality in any disease, particularly in a disease such as Tuberculosis, deaths from which might quite easily be attributed to other conditions such as fever, bronchitis, pneumonia, diarrhoea, and dysentery. We have consequently to rely on statistics such as they are, for an idea of the mortality from this disease. The Sanitary Commissioners' reports contain figures showing mortality from this disease among troops and jail population, but there are no figures for the general population of India. But if the disease is so prevalent in countries like England, Ireland and America, with improved sanitation and less over-crowding, it is safe to assume that it is widely prevalent in India. I believe that this is so and to judge from the figures of mortality in Calcutta quoted by Dr. Kailas Chander Bose in his paper read at the last All-India Sanitary Conference, it appears beyond all doubt that the disease is exceedingly prevalent in India. I admit that the figures of one town are meagre as evidence, but they are useful as an indication of the prevalence of the disease in most large and crowded towns of India. If we accept this prevalence the matter assumes very great importance and urgency. This opinion is borne out by those who have been in a position to make observations or to gain mental impressions in regard to this disease. From my personal experience of six years and the impressions derived therefrom, I am of opinion that the disease is far more prevalent in India than is generally believed.

Opinions such as these latter based as they are on mental impressions, though not as valuable as actual figures, cannot be ignored entirely and we must accept them as evidence till we have reliable statistics. The question as to how and when we will obtain these figures is one which will depend on what response the public will make to the organisation of measures for dealing with the whole problem of the prevention of tuberculosis. But in the meantime the facts of the prevalence of tuberculosis are serious and therefore urgent, and call for some steps to be taken by the public assisted by Government to lessen or circumscribe the evil. It will be useful to reiterate here the various measures being taken in this connection by most countries which have been awakened to the fact of the danger amongst them. These are briefly:—(1) Notification in order to obtain reliable statistics of prevalence and mortality. (2) Formation of societies by the public for (*a*) the education of the public by the free distribution of literature on the dangers of the disease, precautions to be adopted, etc. (*b*) The visitation of the tuberculous in their homes. (3) Treatment of the tuberculous (*a*) by the establishment of sanatoria for consumptives, (*b*) establishment of tuberculin dispensaries. Let us consider in what way could India follow the example of these countries.

1. *With regard to notification.*—If Tuberculosis were included in the list of notifiable diseases, it would be a very great step in the campaign against the disease, for then we would have reliable facts and figures for the information of the public, and due appreciation of these figures would soon result in an awakening of public opinion as to the danger in their midst, and a consequent desire for measures for minimising the evil. Bombay, I am glad to see, has adopted the

notification of Tuberculosis, and if this can be done by one municipality why can it not be applied generally? There appears to be no objection at all provided it does not lead to the inconvenience or petty persecution of those suffering from the disease. There need be no fear of this if the registration of cases were kept strictly under the supervision of responsible officers and treated, as in other countries, as *confidential*. This would prevent the possibility of anything in the way of annoyance of those who were "notified" as suffering from the disease; and as notification would not entail segregation it would make all the difference to the comfort and peace of mind of the patient and relatives. I believe it would be quite feasible in India and I would strongly recommend it as an important step in our progress. But the public should ask for it through their representatives before the idea could be entertained.

2. Next in importance in my opinion is the *formation of anti-tuberculosis societies by the public*. As on these will be depend the success of any measure adopted for the welfare of the public, their formation is a matter that should be seriously considered beforehand and a comprehensive scheme drawn up. Unless this is done at the outset minor societies will be coming into existence with their different forms and rules of association, making it difficult to effect co-operation. These societies would be formed of public men, but if they are to have the sympathy and help of Government, which is essential, then Government should be represented in the person of one or more medical officials on the board of direction. Without this, progress would be very slow and difficult. We have the case of a most useful society at present inaugurated by the efforts of the late Mr. Malabari—a society which is doing valuable work and which might do even still more valuable work. One can't help but feel that without proper direction and advice such societies may be uselessly frittering away time and money in certain directions which might be better employed in some more useful channels. To obviate these drawbacks and to unify the policy of all such societies, I would suggest that a Central Tuberculosis Society be established with a representative board of directors from the various provinces; presided over by the head of the medical service with provincial branch societies and also town committees. It would be necessary to establish central and provincial bureaux officered by those with experience of tuberculosis. The central bureau would be responsible for the collection of facts and figures on Tuberculosis and all literature connected therewith and the printing and distribution of this information in a suitable manner for the use of the public, to the provincial bureaux, whose duty it would be to pass them on to the town committees for distribution to the public. The town committees and provincial bureaux would reciprocate by furnishing figures and statistics on prevalence, treatments, etc., together with periodical reports to the Central Bureau. The advantages of one general association with such branches would ensure a common line of policy being adopted and worked out, and this would lead to much more effective measures being adopted for the conquest of tuberculosis, particularly if Government sympathy and help is assured in the way indicated. The details of constitution of societies would be worked out later, but the objects would be more or less (*i*) Education of the public by, (*a*) distribution of literature in various languages instructing them in the infectivity of the disease, and how to avoid and prevent it, (*b*) visitation of patients to give personal instruction on preventive and curative measures, (*ii*) the provision and support of sanatoria and tuberculin dispensaries for the tuberculous.

3. *Treatment of the Tuberculous.*—(*a*) By the establishment of sanatoria. This appears to have been accepted as the "end all" of efforts for the eradication of tuberculosis. It is certainly necessary to have sanatoria for the accommodation of consumptives, but we cannot thereby hope to deal with even more than a very small fraction of the persons suffering from tuberculosis. If we had a sanatorium for every town in India we might hope to cope with the evil, but sanatoria are expensive and their utility is limited, owing to the small number of patients admissible and the cost of accommodation. The average cost of maintenance of one patient in a sanatorium in Great Britain works out to something between £30 to £50 per annum. By all means have sanatoria but do not let us deceive ourselves into believing that we have then dealt effectively with the whole question of treatment of the tuberculous. Sanatoria in the hills are excellent but they

partake of the nature of a luxury and cannot be taken advantage of by the great majority. If sanatoria are to be built it must be recognised that India is not yet prepared to accept them on the same plan as those established in European countries. The purdah system though gradually disappearing is still observed in many households and must be allowed for. India is not yet sufficiently Europeanised to allow of a daughter or wife being left in a room in a sanatorium, perhaps miles away from her home, without female relations to look after her. So that any plans for sanatoria must take into account the accommodation for relatives who come with the patient; and rooms for the relatives should for obvious reasons never be occupied by patients. Sanatoria should be so arranged that the female patients would be accommodated apart from the males, and the former attended by women only. If this were done it would make sanatorium treatment popular. A plan of model sanatorium cottages should allow for a room and bath room for the patient, and a room, bath room and outside kitchen for the members of the family, with a wide verandah; and if the cottages were separated so that males were on one side and females on the other, it would allow for the privacy which is absolutely essential. Such cottages if built of light material with patent stone floors should not cost more than 600 to 800 rupees each, furnished. On the other hand, there is no reason why each city should not have model sanitary cottages erected just outside the town by public spirited men to accommodate consumptives who are fairly advanced. The poor would be accommodated free, while those who could afford it would pay rent. Such cottages could very well be built near the tuberculin dispensary for the town, and medical aid would then be available. Sanatoria cannot for obvious reasons admit those in an advanced stage. This restricts the value of these institutions for the amelioration of the disease in general.

(*b*) Treatment of the tuberculous by establishment of tuberculin dispensaries. We have in tuberculin, if carefully and judiciously used, an unequalled remedy for the treatment of tuberculosis. Its discovery and introduction by Koch, the expectations it first aroused and the subsequent disappointment owing to its apparent failure, and its re-instatement, are all ancient history. Its apparent failure was due in most instances to its misapplication, in cases of mixed infection or in too large doses. To Sir Almroth Wright is due the introduction of the theraputic use of tuberculin by the application of his opsonic method, by which he was enabled to estimate the results of inoculation of tuberculin and graduate and interspace his poses thereby. We are now receiving abundant evidence of the good results of tuberculin in the treatment of tuberculosis from various workers in different countries. The general impression which is forced on one by the results recorded in various reports as well as one's own experience, is that Tuberculin is a valuable remedy for Tuberculosis and that it should be very generally but circumspectly applied. So much for tuberculin as a curative agent. It is also considered particularly valuable in the diagnosis of tuberculosis, especially in those early cases which offer the greatest hope of success if discovered in time and treated with tuberculin. Such cases would otherwise go undetected till they had passed into the second or third stage and would then have to seek sanatorium treatment. Its value in this respect and as a valuable aid in the campaign against tuberculosis cannot be overestimated. The results obtained with tuberculin in treatment and diagnosis amply justify the extension of its use in such manner as to bring it within the reach of the poorest. The artisan or labourer cannot afford to leave his work and seek treatment in sanatoria. He must continue to earn his wage till the disease is so far advanced that he is eventually compelled to take to bed. This means not only the cessation of the wage but increased expense for medical treatment of the bread winner. I have seen instances of this over and over again during the past six years. But if, in the early stages when tuberculin offers the surest hope of renewed health and strength, he were able to undergo treatment in his own city, it would not be necessary for him to leave his employment for a day. And this will only be rendered possible by the establishment of tuberculin dispensaries in every city. Dispensaries are much less expensive than sanatoria. A municipal tuberculin dispensary has been established recently at Portsmouth, and Dr. Fraser, * who is in charge, states that the sum of £600 per annum would pay for a dispensary and everything connected

* Report of Sanatorium and Tuberculin treatment, Portsmouth Municipal Tuberculin Dispensary by A. Mearns Fraser, M.D.

with it, including a nurse, a care-taker, drugs, apparatus, etc., and allowing the dispensary to be open 5 hours a day and 6 days a week, 60 patients could be treated each week. It would in fact cost as much to maintain 6 patients in a sanatorium as to treat 60 patients at a Tuberculin Dispensary. Thus in general utility it is far and away superior to a sanatorium. The medical officer is in a position to advise and treat hundreds of patients at a dispensary, whereas the sanatorium medical officer's advice and treatment are limited to quite a few. Among the hundreds who come to the former, there is the further advantage which is a very important one, of being able to diagnose the very early cases where the patient only requires a little assistance with tuberculin in order to assure health without his having to leave his work or home The medical officer would also come in touch with cases which would otherwise never be notified as suffering from tuberculosis, and in the case of a death from tuberculosis he would be the most likely person to know of it This information would be valuable help towards the preparation of accurate statistics in regard to the prevalence and mortality from Tuberculosis.

As regards *location*. If this dispensary were located near the outskirts of the town, it would ensure the requisite medical supervision of those cases who being in an advanced stage of Tuberculosis were obliged to live in model cottages just outside the town. These cottages would provide the fresh-air treatment necessary, and at the same time be models for imitation by citizens who desired to build dwelling houses, so that in time with the approval and help of the town planning committee, new sanitary towns would spring up composed of sanitary dwelling houses, to take the place of the old insanitary towns, and this would not only mean salvation from tuberculosis but also plague, malaria and other diseases.

As regards the medical officer, it would be a mistake to "ruin the ship for a ha'porth of tar" but this would be the case if a whole-time officer of experience were not appointed. If such men are not available immediately, it would be far better to wait till they are trained rather than appoint cheap substitutes. It is on the knowledge, tact and kindly manner of the medical officers that the success of the movement will depend. It would be false economy to expect an already hardworked Civil Surgeon or Health Officer to do more than exercise supervision. I believe there will soon be no dearth of trained Indian practitioners who would be glad to accept the appointment.

In conclusion I would strongly recommend the establishment of Tuberculin Dispensaries in every town. If anything, they are more suitable in the case of India than in European countries—and it is the form of medical attendance that is already taken great advantage of by the masses. I am convinced that if they are established on the lines indicated they would be extremely popular. In order to bring this about it is necessary that our public men should interest themselves on behalf of the masses and form anti-tuberculosis societies, and if they will do this the Tuberculin Dispensary will soon be an accomplished fact.

ALL-INDIA SANITARY CONFERENCE—MADRAS—NOVEMBER 1912.

REFLECTIONS AND PROPOSALS RESULTING FROM AN ENQUIRY INTO THE CAUSES OF THREE EPIDEMICS OF MALTA FEVER OCCURRING IN THE 37TH LANCERS AND 10TH LANCERS IN INDIA.

BY

Captain T. F. Paterson, I. M. S., and Captain H. C. Brown, I. M. S.

One of the best pieces of work done by any commission of enquiry into disease was that of the Royal Commission on Malta Fever. This commission not only succeeded in marking down the proximate cause of the disease, but by its suggestions, which were followed out, succeeded in eradicating the disease from an important section of the population in the Island of Malta—the garrison.

Malta Fever, a name which has now become altogether a misnomer, seems to present very much the same characteristics in India as it does in Malta. Comparatively little attention, however, seems to have been paid to the disease and we are far from knowing at present anything of its true prevalence. Our knowledge of its epidemiology remains practically in the same position where it was left by the Royal Commission, such work as has been done on the subject being of the nature of confirmation of main issues. The mortality rate cannot be said to be known of a disease of which the types and diagnosis are still so ill defined. Attempts at prevention seem to be quite unco-ordinated and of a very sporadic nature. We offer no excuse therefore for raising the question of the delineation and prevention of Malta Fever.

The cases which form the subject matter of this paper follow closely upon one another and deserve the name of epidemics. Of these there are three.

(1) A series of six cases occurring among the 37th Lancers at Lahore Cantonment, with no deaths.

(2) 17 cases in the same regiment and in the same cantonment. All the cases were admitted to hospital within a period of 38 days, namely, 16th April —24th May 1912. There were two deaths.

(3) 24 cases in the 10th Lancers. Of these 15 apparently became infected either on the way to or from or actually at the Delhi Durbar, during December 1911, and the remaining 9 in Jullundur Cantonment.

In each of these epidemics the diagnosis was made on the basis of the clinical picture and of serum agglutination tests.

Now the occurrence of all these cases within short periods may have been accidental or due to a rare concurrence of circumstances, but at all events they seem to justify a consideration of the facts which have issued from our enquiry into the particular cases observed by us, and of any general proposals which may be made with regard to the prophylaxis of the disease in India. This may, we think, best be done by discussing the subject as systematically as possible under certain definite headings.

(1) *Type of disease.*—Investigation in India (Wimberley I. M. G. April 1907 and Brayne, *ibid* December 1907) has shown that this may be very variable, all types of fever, mild, medium, and severe are to be met with. Cases may be ambulatory or require long confinement to bed. What does seem peculiar to the disease is the prolonged nature of the convalescence which may be necessary after what, on the basis of the fever criterion, might be regarded as a mild case. Our own cases bear out these characters, a severe type of fever ending in

death, a moderate undulant type, and mild type. It is the moderate, undulant type which is commonly regarded as characteristic of Malta Fever. Even in the third type here differentiated the patients were left in a very debilitated condition, and for a considerable period after leaving hospital complained of severe pains resembling sciatica and lumbago. Of the ambulatory type we have had little experience.

(2) *Diagnosis of the disease.*—In 1897 at Netley, Wright and Smith found that an agglutination reaction to Malta Fever could be obtained with the blood of certain soldiers invalided from India for Typhoid Fever and Chronic Malaria. The clinical diagnosis was certainly incorrect and the true light was shed on this condition by the result of the serum reaction. Not till 1900 were cases of Malta Fever recorded among Indian Troops. Between 1900 and 1904 several cases were diagnosed on the basis of agglutination reactions. But this period also represents the period during which doubt had arisen as to whether Malta Fever really existed in India at all. In 1904, Lamb and Kesava Pai proved in indubitable fashion that the disease did exist in this country, since when, in every year, cases have been recorded as occurring in the Indian Army as well as amongst the civil population. The appended table I is taken from the Sanitary Commissioner's reports, and gives to some extent a delineation of the course of events in the history of the diagnosis of the disease in India. The diagnosis is now made on clinical and serological grounds, the technique of spleen puncture used by Lamb and Kesava Pai for the purposes of proof not being required for diagnosis.

(3) *Contraction of infection.*—This may be regarded as being mainly, if not certainly due to the ingestion of infected food and indeed of milk. That this is a mode by which the disease is contracted has been abundantly proved by the Malta Fever Commission. Their experiments while by no means excluding other modes of infection, do not suggest their very great likelihood. The experiment of stopping all supply of goat's milk to the Malta garrison, resulting as it did in the disappearance of Malta Fever from amongst them, is perhaps the strongest of all arguments that the vehicle of infection was that milk. If infection by contact was a common occurrence, we should expect that epidemics such as we are discussing would show characteristics such as occurrence of the disease in the occupiers of the same quarter. The distribution of the cases amongst the 37th Lancers gives no indication of any such occurrence, rather does the distribution suggest some outside source of infection, we look therefore to milk in some form as the likliest cause of infection in India as in Malta.

(4) *The carrier.*—Owing to the researches of Koch upon Typhoid fever we are more familiar at the present time with the carrier problem than were the Malta Fever Commission. The importance of this side of the question cannot be overrated. With agreement on the subject of infected milk as the cause of this disease, we are supplied with one definite link in the epidemiological chain. If we agree further as to the animal or animals supplying the milk, we shall have still another link; and as the strength of a chain lies in its weakest link or links, so will our prophylactic measures be the more effective the more of these links and the more knowledge of them we may obtain.

The Malta Fever Commission laid the greatest stress upon the goat as carrier, but did not exclude the possibility of other animals being concerned in the matter. Now the goat is in India, as in Malta, a large supplier of milk, but other animals such as cows and buffaloes must also be considered and so we require to examine into the evidence for a particular carrier.

(5) *Differentiation of carrier.*—Now the goat in India has been not only incriminated as a carrier but has been proved to be so. This was done by Forster in 1906 on the basis of agglutination reactions of the blood serum of a herd of goats supplying milk to a regiment which had numerous cases of Malta Fever. The actual organism itself was isolated from the milk of two goats suspected to be suffering from infection by Lamb and Kesava Pai. But before concluding that the goat is the only animal with which we are concerned in prophylaxis, it will be well at least to seek for negative instances to this positive finding. The regiments in which our epidemics occurred were cavalry regiments,

we may therefore enquire whether the horse may not be the hidden enemy dealing disease. To this end we examined for serum agglutination in the blood of a considerable number of horses. The Malta Fever Commission did conclude as to the actuality of the horse as a carrier—(Report, part 6, page 105) We examined 351 horses, some suffering from "Fever" and some from no evident affection. The results of our agglutination tests are given in the tables 2 and 3.

We found no relation between cases of "Fever" and agglutination, nor does the grading of the titres in which agglutination occurred suggest anything but a grading of naturally occurring agglutinations. The agglutinations are certainly higher than those given normally by human beings as recorded by Mr. Fox in his paper, "The variability of agglutination of B. typhosus and M. melitensis by normal sera and its importance in laboratory diagnosis."—Lancet, September 23rd, 1911—but in many ways normal horse serum gives higher immune reactions than other animals. We cannot further find evidence from an examination of our cases that those who were specially concerned with grooming and attending to the horses showed any greater incidence rate than those who did not. In the one regiment the syces did most of the grooming, in the other it was the sowar who groomed and the syces went out to gather grass. We consider, therefore, that there is no evidence whatever to incriminate the horse as a carrier.

If now goat's milk be regarded as the only vehicle of infection, we may seek to find our negative instance by endeavouring to obtain evidence of the occurrence of Malta Fever in individuals who drink no goat's milk. We enquired into this point carefully in the case of the 37th Lancers; in the matter of drinking goat's milk apparently considerable differences occur amongst different races of Indians. This we found to be the case amongst the Awans, Tiwanas, Pathans, etc., composing the regiment. As these races are divided up in definite squadrons it became interesting to note what was the incidence of Malta Fever in the respective squadrons. We took for purposes of illustration ten men per squadron, and table 4 gives the result of our interrogations. This table is to be compared with the representation of the incidence of Malta Fever by squadrons. B. squadron shows no Malta Fever cases, and yet the men of this squadron seem to be those showing a preference for unboiled milk and the milk of outside vendors and of goats. The reverse argument too might be adduced that those who only drink their milk boiled or who deny the use of outside goat's milk have cases of occurrence of Malta Fever in their squadron. However, we do not lay a great deal of stress on this argument in view of the small number of cases investigated and in view of the vagueness of some of the replies. The disproof, if we can call it so, of causation of goat's milk has its value still further diminished by an examination of the interrogatory applied to the cases of Malta Fever themselves. Here we find that with one exception all confess to having drunk milk from the herd of goats which their owner was in the habit of driving past the lines. Some of the answers are qualified as to the state in which the milk was drunk, boiled or unboiled, but that we need not consider as any specially valuable negation of our proof. The Malta Fever Commission's report also contains this type of negative evidence—(Colonel Davis, Part 4, section 13), but the fact still remains that Malta Fever was eradicated from the garrison with the banishment of the goat, and therefore we may say that the onus of proof of another carrier lies entirely with our assumed objector to the more definite proposition. We may take in our endeavour to find this proof the case of two other common animal suppliers of milk in India, the cow and the buffaloe. We had occasion in our investigation to examine the blood and milk of a cow belonging to one of the Native Officers of the 37th Lancers: this animal was emaciated and giving serous milk. On examination of the blood of this cow we obtained with the serum complete agglutinations in a dilution of 1 in 400, whilst with the whey of the milk an agglutination of 1 in 32 was obtained; both these agglutinations were quite unusual and betoken, in our opinion, this animal as a carrier of Malta Fever. The completion of the evidence would have been given had we been able to isolate the Malta Fever organism from the milk. This we were unable to do in a specimen forwarded to the Central Research Institute laboratory, to which a minimum quantity of chloroform had been added. We simultaneously examined the whey from seven other samples of cow's and

buffaloe's milk supplied to the regiment, but none of these gave a positive agglutination even in two-fold dilution.

The evidence here given is strongly in favour of the possibility of the cow being a Malta Fever carrier and transmitter of the disease. Additional evidence is perhaps afforded in this direction by the cases of Europeans admitted to the station hospital with Malta Fever. These men deny ever having drunk anything but cow's or buffaloe's milk, and a Government supply certainly does not include a ration of goat's milk. We had an opportunity of examining the blood of 20 cows and 20 buffaloes from the Government Dairy, Lahore, with the result shown in Table 5.

The results although very definite in three cases with agglutination of 1 in 8, do not warrant any deduction as to the presence of Malta Fever in these animals. There is however not much likelihood of such a carrier, excreting the organism in the milk, continuing to remain for long in a Government herd.

We have then only obtained evidence of infected milk in other animals than the goat in one case. The point is worthy of further investigation.

In this discussion we undoubtedly lay great stress on the agglutination reaction. At the Central Research and Pasteur Institutes, Kasauli, where these tests were done, particular care is taken to control all agglutination tests for Malta Fever. Details of work on this point are given in an article by Mr. Fox of the Pasteur Institute in the Lancet, September 23rd, 1911.

(6) *Mode of development of infection in the carrier.*—Let us suppose that we have settled the point as to which animal is the carrier, we may now ask ourselves in our endeavour to have the complete chain of causation of this disease, how Malta Fever is maintained in that carrier. How does the goat get its Malta Fever and how is the disease kept up? Various suggestions present themselves. Malta Fever organisms are we know excreted in the urine, goats are huddled together in stables and the inoculation of abrasions by urine-infected litter or the inhalation of infected dust are by no means impossibilities. The evidence of the Malta Fever Commission on these counts affords some, although not absolute proof of this. Another possibility is that the continuance of infection is dependent on transmission by milk from mother to kid.

We have no extended experiments of our own to present on this point. We have, however, continuously observed the effect of suckling on a kid by a goat artificially infected with Malta Fever.

The course of events is interesting both as regards the mother and the kid. The mother was inoculated subcutaneously with the contents of six agar culture-tubes of Malta Fever incubated for six days on 5th August 1912, and again with three and one half similar tubes on 4th September 1912. The results obtained by the agglutination tests are given in Table 6, in which is shown the correlation between the agglutinations of the whey and milk from day to day.

This table teaches us that the milk as compared with blood is slow to respond by development of agglutinating power to infection with Malta Fever. The variations too which occur in the agglutinating powers of blood and milk, respectively, do not seem, from a study of this single case, to run exactly parallel. We shall refer again to these reactions under the head of practical tests for carriers.

We may say that the causes of development of Malta Fever in the carrier, the duration of the infection, the stage at which it becomes dangerous to man, and the effect of the infection on the animal itself are all points still requiring further elucidation.

(7). *Practical tests for carriers.*—The tests so far applicable are the blood examination test, the examination of milk for agglutinins, and the isolation of the organism in the milk itself, of these the blood examination test is not always easy to apply owing to prejudice against removal of blood. The occurrence of agglutinins in milk is a late phenomenon, and not always present in marked

degree. The isolation of the organism from the milk is a procedure requiring considerable technical skill and is scarcely to be regarded as offering much hope of a practical solution of the difficulty of isolating the carrier.

If a distinctly selective medium for the isolation of the organism from milk could be compounded, the procedure would be rendered less difficult. We have recently tried the effect of using a high-titre serum on naturally infected milk and certainly had no difficulty in isolating the Malta Fever organism from the deposit which formed. Then, too, it is possible that some form of cutaneous or ophthalmic test might enable us to differentiate the carrier from the non-carrier. We are experimenting in this direction likewise.

(8) *Modes of prevention of Malta Fever.*—The vehicle of infection for man being known, the carrier defined, and the cause for maintenance in the carrier discovered, we should be in possession at once of modes of prevention. The first two are practically known, the third requires investigation.

(*a*) Prevention applied to the vehicle of infection. This means sterilisation of the milk as regards the Malta Fever organism. Dalton and Eyre found that the Micrococcus melitensis was killed by an exposure of 57·5°C. for 10 minutes. They did not make their test with milk. Rosenau found that in milk most of the micrococci die at a temperature of 58°C. and all at 60°C. He concludes that a temperature of 60°C. maintained for 20 minutes is more than enough for sterilisation.

We tested the point in the case of naturally infected milk by heating the milk up to certain temperatures and then allowing it to cool at room temperature for 30 minutes with the result shown in the appended table.

It may not always be practicable to carry out the sterilisation oneself, and therefore it would be as well to have some easily applied test which should afford evidence of the thoroughness of the procedure. The inspecting officer testing the milk ration for troops would find such a test very useful. There are many indicators which may be used for the purpose. Most of them depend in their action on the presence or absence of peroxydases; these ferments, ordinarily present in milk, are destroyed at comparatively low temperatures. Again we must be prepared to detect the addition of unboiled milk to boiled. The test which we have chosen and which seems to us to be the most practicable is Soul's Ortol test.

A quantity (10 c. c) of milk was raised in test tubes to temperatures of 53, 63, 73, 83, and 93°C. and then cooled rapidly. The temperature of 93°C. is the boiling point of water at the altitude of Kasauli (6,000 feet). In this test a dilute solution of Ortol in distilled water is made up fresh. A volume of this solution equal to that of the milk is added to each sample to be examined, and then a few drops, usually three, of Peroxide of Hydrogen. The result in the above experiment was that the milk which had been heated to 93°C remained white, that heated to 83°C became pink, and those heated to lower temperatures became instantaneously brick-red.

As regards the addition of fresh milk to boiled milk, we found that a faint reaction could be obtained with the addition of one part fresh milk to nine parts boiled.

(9) *Prevention applied to the carrier.*—This means the elimination of the carrier from the supplying herd or the requirement of a certificate of immunity from any animal supplying milk. The tests which have been described in this paper would be those to be applied here, in conjunction of course with an inspection of animal or animals for the outward signs of infection. The special tests are essentially laboratory tests, and we have sought for a method by which these could be carried out even in a laboratory situated at some distance, a very common circumstance in India. We are still experimenting with this object in view, but we have found a possible means of getting over the difficulty in the use of chloroform. A milk, the whey of which gave an agglutination up to 1 in 32 with the Micrococcus melitensis was shaken up with chloroform (1 part with 36 of milk) and corked. It was found that this addition of chloro-

form did not interfere with the coagulation of the milk by rennet necessary to obtain the whey, and when tested on the 2nd, 5th, 7th, and 13th days the whey of this chloroformed milk showed no change in the agglutination reaction. In obtaining the whey a minimal amount of rennet should be used as an excess as a restraining influence on agglutination. We used Cross and Blackwell's essence of rennet, and found that when one volume of this was added to sixty volumes of milk, the whey gave a positive agglutination of 1 in 32, whereas when 5 and 10 volumes of rennet were added an agglutination of only 1 in 8 was obtained in each case.

The addition of chloroform in excess to milk will result in complete sterilisation, but short of this a point may be found at which only inhibition of multiplication of organisms takes place. If this is so, it may be possible to send samples of milk to a distant laboratory not only for the application of the agglutination test, but also for the recovery of the M. melitensis if present. We added one part of chloroform to 180 parts of naturally infected milk and were able to recover the micrococcus by plating on ordinary Agar 9 days later. We have also experimented with high-titre sera to see whether a specific agglutination of the Malta Fever organism in a milk containing it could not be brought about in this way. Such a method might greatly facilitate the recovery of the micrococcus in a contaminated milk if it worked out satisfactorily as in the following experiment:—1 volume of milk and 1 volume of high-titre serum gave a sediment after incubation in a sedimentation capillary tube for 1 hour, inoculation of one loopful of sediment on an Agar slope afforded colonies of M. melitensis in pure culture.

We have insisted especially on these tests for the detection of the carrier as applied to the milk itself, because of their ease of application in a laboratory and because of possible difficulties in the way of obtaining blood from an animal. But when all is said and done, it must be confessed that the serum agglutination test is still the most effective means of diagnosis in our hands. We have hopes of improving the other tests and also of experimenting further with cutaneous and ophthalmic reactions.

In conclusion we may set down our ideas as to what might form fit subjects of enquiry in this disease.

(8) *Subject for enquiry.*

(1) Degree of prevalence of the disease in man.

(2) Differentiation of carriers.

(3) Elaboration of tests for carriers.

(4) Exclusion of other modes of infection in man than by ingestion of milk.

(5) Prophylactic measures.

(6). Remedial measures, particularly the use of vaccines therapeutically.

Finally we wish to express our thanks to Major W. F. Harvey, I M S., Offg. Director, Central Research Institute, Kasauli, for his help, also to the Officer Commanding, 10th Lancers, for kindly allowing us to examine the horses of his regiment, and to Lieutenant Cunningham, R. F. A., for having taken and forwarded to us more than 200 samples of horses blood for examination.

TABLE I.

Statement showing the incidence of Mediterranean Fever in the Indian Army from 1900 to 1911.

Year.	Number of cases.	Number of deaths.
1900	7	*Nil*
1901	6	*Nil*
1902	4	*Nil*
1903	8	*Nil*
1904	5	*Nil*
1905	43	1
1906	38	1
1907	62	2
1908	23	*Nil*
1909	13	2
1910	23	1
1911	24	3

NOTE.—Showing the stations in which Mediterranean Fever occurred.

1900.

Cases reported from Meean Mir, Peshawar, Datta Khel and the Malakand Field Force.

1901.

Cases reported from Ambala and Jhansi were confirmed at the Pasteur Institute, Kasauli.

1902.

Two cases at Fort Lockhart.

1904.

Case reported from Kila Drosh, Agra, and Rawalpindi (3). These latter were diagnosed by the agglutination test.

1905.

Cases reported from Rawalpindi (17, of which 12 were Sikhs), Ferozepore (14), Multan (10) and Delhi and Amballa.

1906.

Cases reported from Ferozepore (22), Jhansi (4), Lahore Cantonment (4), Jullundur (3), Ambala, Delhi, Agra, Meerut and Edwardesabad. Of the 38 cases reported in this year 32 were Sikhs.

1907.

Cases reported from Rawalpindi (30, of which 28 were in one Regiment), Jullundur (7), Lahore Cantonment (4), Bannu (4), Ambala (5), Multan, Meerut, Kohat, Secunderabad, Fort Sandeman, Fyzabad and Sialkote.

1908.

Cases reported from Ambala (11), Sehore (2), Sialkote, Rawal Pindi, Dera Ismail Khan, Multan, Jhansi, Kila Drosh, Chitral and Fort Lockhart.

1909.

Cases reported from Dera Ismail Khan (7), Sialkote (4), Kila Drosh and Quetta.

1910.

Cases reported from Peshawar (5), Lahore Cantonment (3), Jacobabad (3) and Nowshera (2).

1911.

Cases reported from Delhi Durbar (9), Lahore Cantonment (6), Kohat (3), Poona (2), Jhansi, Bolarum, Secunderabad and Multan.

TABLE 2.

Showing the result of agglutination tests M. melitensis obtained with blood serum of horses from 3rd Ammunition Column, Royal Field Artillery, and 10th Lancers.

Dilutions Serum	0	4	8	16	32	64	Total.
Frequency	12	54	138	44	8	*Nil*	256

TABLE 3.

Dilutions Serum	0	10	20	40	Total.
Frequency	36	41	16	2	95

Grand Total ... = 351
of horses examined.

TABLE 4.

Statement showing answers received from 10 men taken by chance in each troop—37th Lancers.

Composition of the troops:—R.-A. L.-A. Jat Sikhs.
R.-B. Awans.
L.-B. Tiwanas.
R.-C. L.-C. Beloochis.
R.-D. Yusufzais Mohmands.
L.-D. Khalils, Bangashis, Afridis.

Sources of Milk supply.

1. Regimental Bazaar Halwai. His milk is supposed to be always boiled before issue. It is a mixed supply of Buffaloes' and Cows' milk.

2. R. A. Bazaar Halwai. Doubtful Supply. Probably rarely boiled.

3. Goat-herd who brought his goats past the lines every evening.

Name of Troops.	Do they like milk?	Do they drink it unboiled?	Do they ever drink milk in the surrounding villages?	Do they ever drink milk from the R. A. Bazaar?	Have they ever drunk milk obtained from the goat-herd, source 3?
R.-A. ...	Yes	Sometimes ...	No	No	In the cold weather, milk obtained from this man was drunk by 4 out of the 10 men.
L.-A. ...	Yes	Very seldom ...	No	No	The 10 men asked, all acknowledged drinking this milk but never unboiled.
R.-B. ...	Yes	Very seldom ...	Sometimes on parades.	One out of the 10 did so.	Four out of the 10 used this milk unboiled.
L.-B. ...	Yes	Yes, frequently...	3 out of 10 said frequently.	1 out of 10 did so	Six out of 10 used this milk unboiled.
R.-C. ...	Yes	No	No	No	No. (The truth of these answers is doubtful).
L.-C. ...	Yes, but only when boiled.	No, but they do so in their villages.	Sometimes on parades and manœuvres.	No	No.
R.-D. ...	Don't much care for it and only when boiled.	Don't drink it unboiled.	No	No	Yes three out of 10 did but always boiled.
L.-D. ...	Don't care much for it and only when boiled.	Don't drink it unboiled.	No	No	Yes seven out of the 10 did but always boiled.

TABLE 5.

Showing the result of agglutination tests Mmelitensis with blood serum of cows and buffaloes from Government Dairy, Lahore Cantonment.

I.—Cows.

Dilution Serum	0	4	8	16	32	Total.
Frequency	19	0	1	0	0	20

II.—Buffaloes.

Dilution Serum	0	4	8	16	32	Total.
Frequency	16	2	2	0	0	20

TABLE 6.

History of observations on the inoculation of live Mmelitensis in the female milch goat.

Date.	Temperature.	Blood Agglutination.	Whey Agglutination.	Date.	Temperature.	Blood Agglutination.	Whey Agglutination.
4th August 1912	102'2	2	0	9th September 1912 ...	103'5	600	8
5th ditto	*103	2	0	10th ditto ...	104'7	400	6
6th ditto	103'4	2	0	11th ditto ...	101'3	400	8
7th ditto	104'5	2	0	12th ditto ...	101'6	200	16
8th ditto	104'6	2	0	13th ditto ...	101'8	600	16
9th ditto	103	16	0	14th ditto ...	101'6	800	16
10th ditto	104'1	40	0	15th ditto ...	101'4	400	16
11th ditto	104'4	80	0	16th ditto ...	103'1	400	16
12th ditto	102'7	120	2	17th ditto ...	102'4	600	32
13th ditto	102'8	300	2	18th ditto ...	103'6	600	32
14th ditto	102'6	400	2	19th ditto ...	101'8	800	32
15th ditto	101'9	400	2	20th ditto ...	100'8	1000	32
16th ditto	101'8	400	4	21st ditto ...	101'9	1000	32
17th ditto	101'6	400	4	22nd ditto ...	101'3	800	64
18th ditto	100'9	800	4	23rd ditto ...	103'1	800	64
19th ditto	103'6	800	4	24th ditto ...	102'8	1400	64
20th ditto	102'8	1000	8	25th ditto ...	102'6	1200	64
21st ditto	103'6	1400	4	26th ditto ...	102'4	1200	32
22nd ditto	102'4	1200	4	27th ditto ...	102'4	600	64
23rd ditto	101'6	800	8	28th ditto ...	102'6	800	64
24th ditto	101'2	800	8	29th ditto ...	103'5	1000	128
25th ditto	101'8	800	8	30th ditto ...	104'6	1200	256
26th ditto	101'6	1000	8	1st October 1912... ...	103'4	800	128
27th ditto	101'6	1200	8	2nd ditto	103'5	1400	64
28th ditto	101'6	1400	8	3rd ditto	103'2	1000	64
29th ditto	100'6	1200	8	4th ditto	102'9	600	64
30th ditto	102'6	1000	4	5th ditto	104'3	600	32
31st ditto	102'1	800	8	6th ditto	102'6	1200	128
1st September 1912 ...	101'6	600	4	7th ditto	102'2	800	32
2nd ditto ...	101'6	400	4	8th ditto	102'1	1200	32
3rd ditto ...	101'6	400	4	9th ditto	101'9	1200	32
4th ditto ...	†103'3	600	4	10th ditto	100'6	1200	32
5th ditto ...	‡105'1	200	4	11th ditto	103'4	1200	32
6th ditto ...	105'6	400	8	12th ditto	103'1	1200	64
7th ditto ...	103'6	600	8	13th ditto	102'8	1400	16
8th ditto ...	103'4	400	8	14th ditto	103'2	600	64

* Inoculated with 6 Agar Tubes of M.m.

† Inoculated with 3½ Agar Tubes.

‡ M. M. first isolated from the milk.

ALL-INDIA SANITARY CONFERENCE—MADRAS, NOVEMBER 1912.

ENTERIC FEVER IN INDIA.

*(An account to date of the results obtained from the practical working of the recommendations of the Enteric Fever Research Committee of the Government of India (1906-1908).**

BY

Major E. D. W. Greig, M.D., D. Sc., I.M.S., On Special Duty for the Cholera Enquiry.

As it is now nearly five years since the Enquiry on Enteric Fever of the Government of India completed its labours, it may be instructive and profitable to give a connected account of the results of the practical working of its main recommendations, more particularly as the subject of Cholera, the problems of which in many respects resemble those of Enteric fever, is being studied at present under the auspices of the Indian Research Fund Association. Before, however, taking up the thread of the narrative from the date of the cessation of the investigations, it will be convenient here to recall shortly the main points in the history of the research on Enteric fever in India.

It was estimated that Enteric fever in India was responsible for an annual loss of £230,000.(*a*) Hence if looked at from the commercial point of view alone it was a subject which called for the most careful investigation. Further it was then shown that,(*b*) "in spite of vigorous sanitary measures, a very real increase of the disease was taking place."

Accordingly the Government of India in 1905 decided to undertake an enquiry into the causation of Enteric fever amongst the troops in India. I was selected by the Government of India, and deputed by the Secretary of State for India, in September 1905, to proceed to Germany to study the methods employed by the German Government in the campaign against Enteric with a view to conducting an enquiry on the subject on my return to India. I worked in Germany at the Keonig. Pr. Institute für Infectionskrankheiten, at Berlin, with Geheimrat Prof. Frosch. I studied the problems also at the various special Institutes in Alsace Lorraine, where investigations on "Enteric carriers" were being conducted on a large scale. The results of my investigations were incorporated in a Report to the Secretary of State for India[3].

On my return to India in February 1906, I was placed on special duty by the Government of India to carry out an investigation on the causation of Enteric fever amongst the troops. Up to this date no scientific research had been made in India on the question of the Enteric "carrier." This enquiry lasted for two years (1906-08); and during this period, working on the lines elaborated by me in my first report,(*c*) my colleagues and I carried out a large amount of research work on the etiology of Enteric, and, for the first time in India, the presence of Enteric "carriers" amongst the troops was demonstrated as well as their significance in relation to the causation of epidemics of the disease. The results of this investigation were published by the Government of India at the end of the enquiry.(*d*)*

(*a*) Enteric fever in India. Roberts—Thacker, Spink, Calcutta, 1906.

[3] *Ibid* 1906.

(*b*) Reports on the Methods employed in the campaign against Typhoid fever in Germany. Also Jour. R.A.M.C. Volume VI, February 1906.

(*c*) *Ibid.*

(*d*) Scientific Memoirs of the Government of India, No. 32; also see Annual Report of the Sanitary Commissioner with the Government of India for 1906, page 16 onwards.

* The enquiry was carried out in India under the direction of Lieutenant-Colonel D. Semple, R.A M.C. (retired), with the assistance of Major E. D. W. Greig, I.M.S., Lieutenant-Colonel Wyville Thomson, I.M.S., Captains D. Harvey, R.A.M.C., F. Norman White, I.M.S., and C. Hodgson, I.M.S., and Assistant Surgeon Paras Ram.

Dr. J. C. G. Ledingham of the Lister Institute of Preventive Medicine, London, in his report to the Local Government Board of England on the Enteric fever "carrier(*a*)" says, in regard to this research, that, it is "perhaps the most thorough bacteriological investigation of typhoid convalescents hitherto reported." Still referring to our investigation he states regarding instances of the infectivity of the "carrier" that it is "the most extensive work which has so far been done in this connection."

One of the main conclusions arrived at from our research work was that the convalescent enteric should be segregated and carefully examined bacteriologically and not allowed to return to his regiment until he has been shown to be free from the B. typhosus.

When the research work ceased, the next phase of the problem was the elaboration of a means of applying the results obtained by our inquiry to the conditions met with in practice in India. This was solved by the Government of India giving effect to our recommendation to establish two Depôts for the segregation and investigation of Enteric convalescents, one at Naini Tal (1908) for the troops of the Northern Army, and later, one at Wellington for the troops of the Southern Army in India. At these Depôts the convalescents are examined, on the lines laid down by our enquiry, by officers of the Royal Army Medical Corps in India, and good work has been done at them by D. Harvey, H. W. Grattan, J. L. Wood, R. W. Clements, J. W. Dawson and others.

I propose to give the results of the working of these Depôts from their commencement to the present date in regard to their effect in reducing the incidence and mortality from Enteric fever amongst the troops in India. I have obtained my information from the various Reports, etc., published by military medical authorities in India. Thus Aldridge(*b*) writes "As regards the effect of this measure on the incidence of Enteric fever at the stations from which the convalescents came, it is found that the admissions for Enteric fever from all stations which sent convalescents to Naini Tal showed a reduction of 9 per cent of the figures for 1907, while the remaining stations show an increase of 26·6 per cent." This is a very significant observation; the stations which did not send their convalescents showed a marked increase of admissions for Enteric and served as a control to the stations which did and which showed a distinct reduction in the admissions.

In the Report(*c*) on the Health of British Troops in India in the year 1910, Colonel Firth, R.A.M.C., the Sanitary Officer at Army Head Quarters in India [now Assistant Director, Medical Services (Sanitary) India], writes, "The really remarkable diminution of Enteric infection among European troops in India during 1910 is difficult to explain by any one condition. The weight of evidence points to the dominant influence of our present-day system of segregation of the infected, with the careful examination of all convalescents and their retention in the convalescent Depôt until found to be absolutely free from infection. It is difficult to appraise too highly the great part played by the two Enteric fever Depôts at Naini Tal and Wellington in controlling the disease." Grattan and Wood(*d*) writing regarding Enteric in India state—

"The incidence of enteric which is becoming less and less each year, points to some condition or conditions at work which have a cumulative effect: we believe that these factors are in order of dominancy:—

1. Segregation of the convalescent enteric patient, until he is proved to be free from infection.
2. The elimination of the "Chronic carrier."
3. Inoculation.
4. General all-round improvement by attention to sanitary details."

(*a*) Report of Local Government Board on Public Health and Medical Subjects (New series) No. 43, 1910.

(*b*) Jour. R.A.M.C., Volume XXIII, September 1910.

(*c*) Report on health of British troops in India in 1910 (quoted in Indian Medical Gazette), December 1911.

(*d*) Jour. R.A.M.C., Volume XVII, 1911.

The following table shows the recent figures regarding enteric fever in the Army in India.(*a*)

Year.	Average admission per 1000.	Constantly sick per 1000.	Deaths per 1000.
1895-1904	22·3	3·31	5·62
1905-1909	13·7	2·75	2·66
1910	4·1	0·91	0·62

Another important result of the investigation was the demonstration by strict scientific methods, that the disease called enteric fever embraced two other infections, produced by the B paratyphosus A., and B paratyphosus B., the former in our experience was the more common.

On May 6th 1906 the B. paratyphosus A was isolated by us (*b*) from the blood of a soldier in the Durham Light Infantry, and, for the first time in India, this disease was separated from enteric fever by strict bacteriological methods.(*c*) Since the discovery opened up the way for further investigation, a number of interesting observations have been made in India, particularly by Harvey and Grattan.(*d*)

Firth(*e*) in a recent paper (September 1912) gives the following table:—

Year.	Number of cases of enteric and paratyphoid fever.	Number of deaths from enteric and paratyphoid fever.	Case mortality percent.
1897	2050	556	27·1
1902	1012	260	25·7
1907	910	192	21·1
1908	448	191	18·8
1909	639	113	17·7
1910	335	46	13·8
1911	274	24	8·8

In 1910, 39 cases, and in 1911, 104 cases of paratyphoid were diagnosed. Hence the figures in the above table, column 2, for 1910 and 1911 may be divided into 296 cases of enteric and 39 cases of paratyphoid for 1910; for 1911 of the total 274 cases only 170 were due to enteric and 104 to paratyphoid.

(*a*) Lancet, December 23, 1911.

(*b*) Scientific Memoirs, Government of India, No. 32. Annual Report of Sanitary Commissioner with the Government of India for 1906, page 16.

(*c*) Scientific Memoirs, Government of India, No. 32. Annual Report of Sanitary Commissioner with the Government of India for 1906. Page 16.

(*d*) Jour. R.A., M.C., Volumes XVI and XVII, 1911.

(*e*) Indian Medical Gazette, September 1912.

It will be seen that although B. paratyphosus A was first cultivated by us (*a*) from the blood of a patient in India in 1906, and the results published, it was not until 1910, 4 years later, that systematic blood cultures were begun amongst the British Troops (*b*) as a means of differentiating this type of fever, but since the commencement of the use of this method of diagnosis the above valuable results have been obtained.

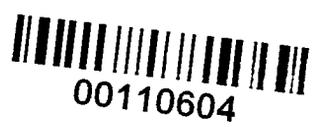
Firth (*c*) states in a recent paper, September 1912, that since the opening of the Enteric Depôts 1,229 cases of enteric fever have been examined and 13 cases of "chronic carriers", and 13 cases of "temporary carriers", have been detected. Also 124 cases of paratyphoid fever have been investigated and one "chronic carrier" and 17 "temporary carriers" have been found. He goes on to say, "Had the old system prevailed and no Enteric Depôts existed, these 26 "Enteric carriers" would have gone back to their barracks at once on discharge from the hospital. The aftermath of cases from these 44 men, assuming that each of them infected but 3 other individuals, amounts by the third generation to as many as 1,188 cases. From this point of view we attach the first importance to the existence of the Enteric Depôts and the elaborate technique there carried out for the detention of potentially infective individuals."

It may be asserted in the light of these results of the working, in actual practice, for a number of years of the recommendations for the prevention of Enteric amongst the troops in India, based on our researches on enteric fever, that, on the whole, the conclusions arrived at in 1907 have stood the test of time fairly well; and that their mortality has not been unduly high; and, what from the practical standpoint, is the most important, that the enquiry was worth, probably, the expenditure in money and time incurred on it.

(*a*) Scientific Memoirs, No. 32.

(*b*) Indian Medical Gazette, 1912, September.

(*c*) Firth *ibid.*



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